Health Physics Vol. 61, No. 3 (September), pp. 317–335, 1991
Printed in the U.S.A.

● *Paper*

CATALOG OF RISKS EXTENDED AND UPDATED

Bernard L. Cohen
Department of Physics and Astronomy, University of Pittsburgh, Pittsburgh, PA 15260

***Abstract*—A large variety of risks are quantified in terms of the loss of life expectancy they cause in the United States. Risks considered include the following: diseases; accidents of various types at home, at work, in public, and in motor vehicles; unemployment; poor social connections; use of small cars; smoking; air pollution; other environmental pollutants leading to cancer and non-cancer effects; purposely ingested substances; sports participation; geography; medical care; epidemics; natural hazards; socioeconomic factors; Rn and other radiation; and energy conservation. A few suggestions for applications of this catalog of risks are offered.**

INTRODUCTION

IN 1979, WE published "A Catalog of Risks" (CR), a review paper in which a large variety of risks were expressed in terms of the loss of life expectancy (LLE) they cause (Cohen and Lee 1979). This was followed by a similar paper on occupational risks (OcR) (Cohen 1981). Our purpose here is to extend and update those papers. The various sections of this paper derive LLE for the types of risks indicated in each section heading.

The advantage of using LLE in quantifying risks is that it is easily understandable in terms of every day experience. For example, a mortality risk of 3×10^{-3} is not as easily understandable to most people as an LLE of 30 d; 30 d of living is something we all experience and can appreciate. Moreover, LLE considers the important fact that a premature death of an elderly person is less regrettable than the death of a young person.

Life expectancy is an interesting concept in its own right, especially with regard to its improvement over time. In the advanced nations of Europe it is estimated that in the 17th century, life expectancy was 35 y in England (Sagan 1987) and 32 y in Breslau (now Wroclaw, Poland) (Urquart and Heilmann 1984). But reliable statistical information was not generally collected until the mid-19th century. Life expectancy was 41.0 y in 1840, 42.5 y in 1860, 45.2 y in 1880, 47.1 y in 1890, 50.5 y in 1900, 54.3 y in 1910, 58.3 y in 1920, 61.7 y in 1930, 64.6 y in 1940, 69.8 y in 1950, 72.0 y in 1960 (United Nations 1973), and is now about 75 y. There is less historical information on technologically underdeveloped nations. An archeological study of an Indian tribe in Ohio indicates that in 950 A.D., life expectancy was about 25 y (Lovejoy et al. 1977). The average life expectancies in 1985–1990 in various countries are listed in Table 1.

(*Manuscript received* 7 *August* 1990; *second revised manuscript received* 16 *April* 1991, *accepted* 30 *April* 1991)

METHODS FOR CALCULATING LLE

Where mortality rates or ratios are available as a function of age, the straightforward method for calculating LLE is by using a life table. Each age range is assigned an index i (i = 1, age 0–1 y; i = 2, age 1–4 y; i = 3, age 5–9 y; etc.) and the probability of dying in that age range, the mortality rate $m(i)$, is obtained from Census statistics as deaths per year per 10^5 people. We start with a cohort of 100,000: the number dying in age range 1, $n(1)$, is then $m(1) \times 100{,}000$, and the number remaining alive after age range 1, $R(1)$, is $100{,}000 - n(1)$. In general,

$$n(i) = R(i-1) \times m(i)$$
$$R(i) = R(i-1) - n(i), \qquad (1)$$

with $R(0) = 100{,}000$. Going back and forth between these two equations for each value of i, all $n(i)$ can be computed. The average number of years lived by those who die in age range i, $y(i)$, is readily estimated (e.g., for age range 25–29, y = 27), and the total number of years they lived is then $n(i) \times y(i)$. The life expectancy for an individual in this population, E, is:

$$E = \frac{1}{100{,}000} \sum n(i) \times y(i). \qquad (2)$$

If a particular risk is removed, the $m(i)$ are changed to $m'(i)$, and a new life expectancy, E', is calculated. Then the LLE due to that risk is:

$$\text{LLE} = E' - E. \qquad (3)$$

Here, the $m(i)$ are taken from 1987 data (U.S. Bureau of the Census 1988). For the entire population, E = 74.9 y.

In some cases, the best information available is that all $m(i)$ are increased by the same amount, r, or by the same factor, f. Numerical calculations like those described

Table 1. Life expectancy (in years) on various continents and in various selected nations in 1985–1990 (World Resources Institute 1987).

World	61.1[a]		
Africa	51.3	Asia	61.1
Algeria	62.5	Afghanistan	39.0
Congo	48.5	Bangladesh	49.6
Egypt	60.6	China	69.4
Ethiopia	41.9	India	57.9
Gambia	37.0	Indonesia	56.0
Ghana	54.0	Iran	59.0
Kenya	55.3	Japan	77.2
Libya	60.8	Korea	69.4
Morocco	60.8	Pakistan	52.1
Nigeria	50.5	Saudi Arabia	63.7
Sierra Leone	36.0	Turkey	64.1
South Africa	55.5	Europe	74.0
North America	72.0	France	75.2
Canada	76.3	Germany (W)	74.5
Haiti	54.7	Italy	75.2
Mexico	67.2	Poland	72.4
United States	75.0	Sweden	76.8
South America	65.5	United Kingdom	74.5
Argentina	70.6	Yugoslavia	71.7
Bolivia	53.1		
Brazil	64.9	U.S.S.R.	72.1
Chile	70.7	Oceana	69.1
Peru	61.4	Australia	75.7
Venezuela	69.7	Papua N.G.	54.0

[a] Calculated from data on continents gives 63.0.

above with eqns (1), (2), and (3) for various r and f lead to results that are approximately described by the empirical relationships:

$$\mathrm{LLE} = 1.1 \times 10^6\, d \times r \quad (r < 10^{-3}) \tag{4}$$

$$\mathrm{LLE} = 13\, y \times \ln f \quad (0.2 < f < 5) \tag{5}$$

with r (expressed in y^{-1}) a dimensionless number. For example, an added risk of 1×10^{-4} y^{-1} at all ages ($r = 10^{-4}$) gives LLE = 110 d, and a risk that multiplies the mortality probability at all ages by 1.5 ($f = 1.5$) gives LLE = 13 $y \times \ln 1.5 = 5.3$ y. If risks are age-dependent, eqns (4) and (5) cannot be used and LLE must be calculated numerically from eqns (1), (2), and (3). Calculations indicate that if the added risks are due to occupational exposure and, therefore, apply only for ages 20–65, the LLE are approximately half of the values calculated from eqns (4) and (5).

The LLE due to a risk can also be understood as the average reduction of lifetime for those involved. For example, if 600 people of an age that would normally have 30 y of remaining life expectancy were to play Russian Roulette (one in six dies instantly), 100 would die losing 30 y of life expectancy, and 500 would not lose any life expectancy. The average LLE for the group would be one-sixth of 30 y, or 5 y, from playing Russian Roulette.

For a risk that causes deaths of N people to occur p years prematurely, where N is a number known from observational experience, we use the fact that there are about 2,000,000 deaths per year in the United States: hence, the fraction of the population affected by this risk is $N/2{,}000{,}000$. Since each victim loses p years, the LLE for the average American is the product of these (cf. the Russian Roulette example):

$$\mathrm{LLE} = pN/2{,}000{,}000. \tag{6}$$

If N is a number estimated from information on health effects due to exposures throughout life, it is interpreted as referring to a life-table age distribution of a population equal to the present U.S. population, in which there would be 3,200,000 deaths per year. With that interpretation, present-day exposures are the relevant factor, whereas if eqn (6) were used, only exposures to a much smaller population long ago would be relevant. With this interpretation, again using the Russian Roulette example,

$$\mathrm{LLE} = pN/3{,}200{,}000. \tag{7}$$

If the information is that a certain percentage, P, of all deaths are due to a risk that costs victims p years of life expectancy, the probability for each person to be a victim is $0.01P$, and as in the Russian Roulette example, the LLE for the average American is the product of these:

$$\mathrm{LLE} = 0.01pP. \tag{8}$$

In both eqns (6) and (7), p could be the *average* number of years by which death comes prematurely.

If information is available on the average age of victims, p may be interpreted as the remaining life expectancy at that age times the fraction of the original cohort still alive at that age. In many cases, p in eqns (6), (7), or (8) must be estimated; in such cases, the uncertainty in LLE is just proportional to the uncertainty in p.

If the data available give the loss of life expectancy (LLE)′ for victims, as in the Russian Roulette example, we need only multiply by the fraction of the whole population that become victims, F, to obtain

$$\mathrm{LLE} = F \times (\mathrm{LLE})'. \tag{9}$$

Uncertainties in LLE are normally determined by the uncertainties in the basic data from which they are calculated. Where these are from national statistics, errors are normally negligible, but where they are from estimates, errors can be a factor of two or more.

DATA FROM DEATH CERTIFICATES

Death certificates give the cause of death as judged by the attending physician. They are the best source of data on deaths from various diseases, but they give little information on risks imposed by human activities or by other readily recognized risk factors. Some LLE from this source (U.S. Bureau of the Census 1990) are listed in Table 2.

Table 2 also lists the ratios of age-adjusted mortality rates from various diseases for male:female (M:F) and for blacks:whites (B:W). It is evident that there are sizeable gender and racial differences in many cases.

Table 2. LLE from various causes of death in the U.S. calculated from the U.S. Bureau of the Census (1990) by use of eqns (1), (2), and (3). M:F = male:female; B:W = black:white.

Disease	LLE (d)	M:F		B:W	
Tuberculosis	4.7	2.0		4.2	
Viral hepatitis	3.3	1.6		1.0	
Cancer—all types	1,247 (3.4 y)	1.21		0.85	
-Lip, oral, pharynx	22	2.14		1.29	
-Digestive organs	269	1.16		0.96	
-Respiratory	343	2.25		0.85	
-Breast	109	0.006		0.78	
-Genital organs	113	1.26		1.17	
-Urinary organs	114	1.86		0.61	
-Leukemia	46	1.29		0.65	
Diabetes	82 (2.7 mo)	0.74		1.45	
Nutrition deficiency	3.50	0.64		1.08	
Cardiovascular	2,043 (5.6 y)	1.02		0.82	
-Heart disease	1,607 (4.4 y)	1.10		0.80	
-Cerebrovascular	250 (8 mo)	0.69		0.96	
-Atherosclerosis	24	0.64		0.58	
Pneumonia	103 (3.3 mo)		1.03		0.76
Influenza	2.3	0.67		0.22	
Chronic pulmonary	164 (5.4 mo)		1.68[a]		0.48
-Bronchitis	7.3	1.29		0.35	
-Emphysema	32	1.95		0.36	
-Asthma	11.3	0.68		1.87	
Ulcer	11.8	0.96		0.71	
Appendicitis	1.2	1.00		2.00	
Liver diseases	81 (2.7 mo)		1.88		1.20
Gallbladder	4.7	0.85		0.62	
Nephritis	41 (1.3 mo)		1.06		1.61
Accidents	365	2.33		1.09	
-Motor vehicle	205	2.61		0.89	
-All other	158	2.08		1.30	
Suicide	115	3.80		0.47	
Homicide	93	3.40		5.70	

In addition to diseases, Table 2 lists accidents, suicides, and homicides. For each, the M:F ratio is greater than 2, and for homicide the B:W ratio is very large. In the following section, more details on accidents will be given. Of the 30,900 suicides in the U.S. in 1986, 59% were with firearms, 15% were by hanging or strangulation, 10% were by ingesting liquid or solid poisons, 10% were by inhaling poisonous gases (82% of these were by motor vehicle exhausts), 1.4% were with knives or razors, and 2.2% were from jumping from high places (NSC 1989). Of the 21,500 homicides, 61% were with firearms, 20% were with knives and razors, and 5% were by strangulation. Between suicides and homicides, firearms caused 31,000 deaths and an LLE of 130 d.

ACCIDENTS

The most obvious and best understood risks are from accidents. Fortunately, these risks have been declining steadily (NSC 1989). In 1906, when the population was 86 million, there were 80,000 people killed in non-motor vehicle accidents, whereas in 1988 when the population was 246 million, only 47,000 died in accidents not involving motor vehicles. This represents a nearly fivefold reduction in mortality rates. Motor vehicle safety has also improved, with the mortality rate now only two-thirds of that in the 1930s. Progress is continuing on all fronts. The number of deaths per 100,000 population declined between 1978 and 1988 from 47.5 to 39.1 (18%) for all

Table 3. Deaths per year and LLE due to various types of accidents for average citizens of U.S. For work-related accidents, LLE are for those exposed 2000 h y^{-1} from ages 18–65 (calculated from NSC 1989).

Accident type	1988 deaths	LLE (d)
All accidents	96,000	366
-Motor vehicle	49,000	207
-Home	22,500	74
-Work	10,600	60
-Public	18,000	60
-Falls	12,000	28
-Poison (solid, liquid)	5,300	20
-Drowning	5,000	24
-Fire, burns	5,000	20
-Suffocation	3,600	28
-Firearms	1,400	6.5
-Poison (gas)	1,000	4.0
-Electric current	850	4.5
-Explosives	250	1.6
-Knives, razors	120	0.7
-Machinery	1,250	6.5
-Falling objects	900	6.0
-Venom plant and animal	60	0.4
-Injury by animals	95	0.6
Motor vehicle	49,000	207
-Collisions	21,200	87
-Non-Collision	13,900	61
-Pedestrians	8,800	36
-Fixed objects	3,300	14
-Pedalcycle	1,100	5.7
-Railroad	600	2.5
Home	22,500	74
-Falls	6,500	13
-Poison (solid, liquid)	4,300	16
-Poison (gas)	600	2.6
-Fire, burns	4,100	17
-Suffocation	2,900	9.1
-Drowning	800	4.2
-Firearms	800	3.8
-All other	2,500	7.9
Public	18,000	60
-Falls	4,100	9.3
-Drowning	2,900	14.0
-Fire, burns	600	2.1
-Firearms	500	2.2
-Air transport	900	3.7
-Water transport	800	3.3
-Railroad	300	1.3
-Other transport	200	0.9
-All other	7,700	22.8
Work (occupation)	10,600	60[a]
-All (114×10^6)	10,600	60
-Agriculture	1,500	320
-Mine, quarry	200	167
-Construction	2,200	227
-Manufacturing	1,100	40
-Transportation, utility	1,400	160
-Trade	1,100	27
-Services	1,500	27
-Government	1,600	60

[a] This column represents risk to workers in that occupation.

accidents, 23.6 to 19.9 (16%) for motor vehicle accidents, 5.9 to 4.3 (27%) for occupational accidents, 10.3 to 9.2 (11%) for accidents in the home, 6.2 to 4.9 (21%) for falls, 3.2 to 2.0 (38%) for drowning, 2.8 to 2.0 (29%) for fire, and from 0.8 to 0.6 (25%) for accidents with firearms. Only for poison has the risk increased, from 1.4 to 2.1 (50%).

The number of deaths in the United States in 1988 and the LLE for the average American due to different types of accidents are listed in Table 3 (NSC 1989). We see from Table 3 that over half of all accident deaths are caused by motor vehicles, 12.5% are caused by falls, and no other cause contributes over 5.5%. Nearly half of non-motor vehicle accidents occur in the home, 38% occur in public, and less than 15% occur at work. The accident deaths per 100 million hours of exposure are seven averaged over a lifetime, five while at work, three at home, four in public (discounting motor vehicle accidents), and 59 in motor vehicles.

About half of all motor vehicle accident deaths are alcohol-related (NSC 1989), and more than half of alcohol-related fatal accidents do not involve another vehicle. In alcohol-related accidents, two-thirds of the victims were under the influence of alcohol and one-third were not. Drivers in fatal accidents are 77% male and 23% female. The male driver fatal accident rate per billion miles of driving is 45 vs. 17 for females. Approximately 18% of those killed are pedestrians.

In 1985–1987, deaths per 1.6×10^9 km (billion passenger miles) were 9.8 in automobiles (down from 12.8 in 1978–1980), 0.3 in buses, 0.6 in railroad passenger trains, and 0.5 in scheduled airlines. Motor vehicle death rates per 1.6×10^9 vehicle km (billion vehicle miles) are 24.6 overall, 16.6 during the day and 41.5 at night, 14.7 in urban areas and 39.1 in rural areas, and 15 on rural interstate highways. When the rural interstate speed limit was increased from 89 to 105 km h^{-1} in 1987 (55 to 65 miles per hour), the above death rates increased from 14 to 16 in the 38 states that implemented the increase. Since 8% of motor vehicle deaths occur on rural interstates, they cause an LLE of (0.08×207 =) 17 d; hence, the increased speed limit gave the average American an LLE of 2 d.

A U.S. Department of Transportation study found that states with seat-belt laws experience 7% fewer fatalities than would be expected without these laws (Partyka 1988). A seat belt law thus increases life expectancy by (0.07×207 =) 14 d. A study in North Carolina (Reinfurt et al. 1988) showed that enforcement of a seat belt law increased usage from 25 to 60% and reduced fatalities by 11.6%. This implies that using a seat belt reduces risk by [11.6% ÷ (0.60 − 0.25) =] 33%, and, therefore, adds (0.33×207 =) 69 d to one's life expectancy. The reduction in injuries was even larger.

The largest contributor to LLE from accidents in the home is fires. Cigarettes caused 29% of fires in homes, 13% were caused by heating equipment, and 12% by electrical problems. A close association between fire risk and poverty has been demonstrated (Fahy and Norton 1989). For example, the annual fire mortality rate per 100,000 is 1.7 nationally, 4.6 for cities with 25% of their population below the poverty level, and 0.64 for those with 10% below the poverty level. Arson is the leading cause in urban poverty areas.

Twenty percent of U.S. homes without smoke detectors experienced 60% of the deaths caused by fires (Hall 1989), which implies that the risk with a detector is only one-sixth of the risk without a detector and one-half of the average risk. Having a smoke detector, therefore, increases life expectancy by ($\frac{1}{2} \times 17$ =) 9 d.

Falls cause the largest number of accidental deaths in the home, but almost 70% of the deaths are among people over 75 y old, which greatly reduces the LLE. This is a factor in several of the accident types, with 37% of all accident deaths in the home being among people over age 75.

Accident deaths in public places are also heavily influenced by the 30% of these among people over 75 y old, including 66% of the deaths from falls. Public accidents also include accidents in public transportation that, as we have seen, are much safer than private motor vehicle transport. However, the number of fatal accidents per 1.6×10^9 aircraft km (million aircraft miles) is much larger when major airlines are not used: 0.24 for major airlines, 3.2 for commuter airlines, 10.1 for air taxis, and 15.8 for general aviation.

A person who flies 400,000 km (250,000 miles) per year (round trip coast-to-coast each week) for 40 y has a 0.5% risk of being killed if he uses scheduled airlines (0.5 per billion passenger miles × 10 million miles), giving him an LLE of ($0.5\% \times 35$ y =) 64 d. [We assume that the average victim loses 35 y of life expectancy and use eqn (9).] If he uses small planes, his risk would be approximately 40 times higher, giving a substantial LLE of about 7 y. The average American travels about 1600 km (1000 miles) per year on scheduled airlines, giving him an LLE of ($64/250 \times 70/40$ =) 0.4 d.

Another interesting breakdown of accidents is by geography. The LLE varies from 740 d in Alaska and 650 d in New Mexico to 250 d in New York. There are 12 states where it is more than 470 d (OR, MT, ID, WY, NV, AZ, NM, TN, MS, AL, SC, AK) and 12 states where it is below 355 d (NH, VT, MA, CT, RI, NY, NJ, OH, MI, MN, UT, HI). The U.S. annual mortality rate from accidents per 100,000 population is 39.5 compared with 23 in England and Japan; 30–35 in Costa Rica, Germany, Sweden, and Australia; 45–50 in Belgium, New Zealand, Poland, and Norway; 50–60 in Switzerland, Austria, and Czechoslovakia; 62 in France; and 73 in Hungary and Cuba. The difference between France and England is enough to cause more than a 1-y difference in life expectancy. If the accident mortality rate in the U.S. were reduced to that of England, our life expectancy would be extended by about 160 d.

OCCUPATIONAL RISKS

In OcR, it was concluded that various types of occupational risk—occupational diseases, stress, and work-

related accidents—give the average American worker an LLE of about 500 d. The new information deals only with accidents, which represent just about 15% of the total.

There are three widely quoted sources of information on occupational accident deaths (most recently reported number in parentheses): National Safety Council—NSC (10,600 in 1988), Bureau of Labor Statistics—BLS (3400 in 1987), and National Institute for Occupational Safety and Health—NIOSH (6400 in 1985) (NSC 1989). Since the latter two are limited by their specific methodologies whereas the National Safety Council attempts to obtain a true estimate, we used the NSC figures. Their breakdown by industries is included in Table 3, updating a similar table in CR.

UNEMPLOYMENT

By far the most dangerous occupation is no occupation—being unemployed. While there is extensive literature on this, including a compilation of review articles (Brenner and Mooney 1983), the most forthright estimate is given by Ray Marshall, former Secretary of Labor,* for a 1% increase in U.S. unemployment for 1 y: 37,000 deaths including 20,200 cardiovascular failures, 500 alcohol-related cirrhoses of the liver, 900 suicides, and 650 homicides. In addition to these deaths, there were 4,200 admissions to mental hospitals and 3,300 admissions to prisons. If all of these deaths were experienced by the unemployed person, he would have a 4% chance of death, which gives him an LLE of about 1.4 y or 500 d. This is roughly one-fifth of the risk of smoking a pack of cigarettes per day throughout adult life, or about equal to the risk of smoking 10 packs per day while unemployed.

Actually, all of this harm is not inflicted on the unemployed worker. It includes effects on his family and friends, and even on those who remain employed but experience stress from fear of unemployment.

OVERWEIGHT

The data on risks of overweight in CR were derived from insurance company data, but insured people are not necessarily representative of the general public. A more recent study was carried out by the American Cancer Society (Lew and Garfinkel 1979) involving 750,000 men and women from the general population. The results are summarized in Table 4 in terms of mortality ratios for males and females. The average of these is converted to LLE using eqn (5), and the last column gives LLE/percent overweight. We see that up to about 30% overweight, this is roughly constant at about 52 d LLE for each percent overweight. If the average weight for a person's height and build is 68 kg (150 lb), each extra 0.68 kg (1.5 lb) gives an LLE of 52 d, or 34 d LLE per 0.45 kg (1 lb.) overweight. This may be compared with 30 d/0.45 kg (1 lb) derived in CR. More details on this problem are given in Table 5, which lists the mortality ratios from various diseases vs. percent overweight.

Table 4. Mortality ratio and LLE vs. percent overweight (Lew and Garfinkel 1979).

Percent overweight	Mortality ratios			LLE (d)	LLE per percent
	Male	Female	Average		
−15	1.05	0.96	1.00	0	—
0	1.00	1.00	1.00	0	—
+15	1.15	1.17	1.16	777	52
+25	1.27	1.29	1.28	303	52
+35	1.46	1.46	1.46	964	56
+45	1.87	1.89	1.88	3,276	73

SOCIAL CONNECTIONS

One of the largest risks in CR was the risk of remaining unmarried, whether single, widowed, or divorced. The LLE is many years, larger for men than for women, and larger for blacks than for whites. In view of the many doubts, questions, and comments received on this particular risk, we give more details here.

The ratios of mortality rates due to various diseases for unmarried to married men and women (NCHS 1970) are listed in Table 6. We see that the risk of being unmarried includes a very wide variety of diseases—cardiovascular diseases, strokes, many types of cancer, accidents, suicides and homicides, and many others.

There have been many studies of health consequences of becoming widowed (NAS 1984). Apparently the risk of death reaches a peak in the second year, and then declines to insignificance after about 6 y. Remarriage removes the risk. There is a marked increase in use of alcohol, tobacco, and drugs following bereavement. Unmarried men are at increased risk following the death of their mothers.

Social connections have important effects on life expectancy (Sagan 1987) in other circumstances. Losing both parents during childhood increases the risk of later suicide by 700% (Chen and Cobb 1960), which corresponds to an LLE = 2.2 y. Losing one parent (by death or divorce) during childhood doubles the risk of later suicide, causing LLE = 115 d according to Table 2, and increases the risk of dying in an accident by 50%, causing LLE = 180 d. It also increases the incidence of several diseases, including tuberculosis and various psychiatric problems.

People with the most social connections have less than half the mortality rates at most ages of those with the least social connections (Berkman and Syme 1979) corresponding to a difference in life expectancy of about 9 y according to eqn (5). We might say that very poor social connections give an LLE of 4.5 y, while very good social connections give an LLE = −4.5 y.

* Marshall, R. Health and unemployment. Paper presented at the American Public Health Association Meeting, Dallas, TX; November 1983. Copies available from author at University of Texas, Austin, TX.

Table 5. Mortality ratios for various diseases vs. percent overweight (Lew and Garfinkel 1979).

Cause of death	Overweight 15%	25%	35%
All causes	1.16	1.28	1.46
Heart diseases	1.23	1.35	1.50
Cancer	1.06	1.14	1.18
Diabetes	1.78	2.95	3.65
Digestive disorder	1.55	1.74	2.54
Cerebral vascular	1.12	1.17	1.47

SMALL CARS

It has long been known and widely advertised that the number of fatalities per year of driving is considerably higher for small cars than for large cars. However, this does not necessarily mean that driving a small car is less safe than driving a large car. For example, it is well known that younger drivers tend to use smaller cars, and perhaps younger drivers are less careful. The distance driven per year, the ratio of highway to city driving, the average speed, the ratio of night to day driving, the ratio of male to female drivers, the probability for the driver to be intoxicated, percent of use on weekend, and the amount of use in hazardous weather are all things that might correlate with car size and hence cause a relationship between car size and fatality rate.

However, there is a tremendous amount of data on motor vehicle accidents from the U.S. Department of Transportation's Fatal Accident Reporting System (FARS) and from various state agencies. By utilizing these in an analysis in which the above-listed factors cancel out in the ratio of collisions of an automobile with a pedestrian, a motorcycle, and another automobile, Evans (1984a,b) finds that a given driver with equivalent conditions is 1.9 times as ... ely to be killed in a 900-kg car as in an 1800-kg car. ...is result is independent of the driver's age. Evans an... Wasielewski (1987) studied collisions between cars o... ual mass. For fatalities and for serious injuries, and f... both head-on and all collisions, they found a consistent ...lationship that the risk increases with decreasing vehicl... ass, doubling as the latter is reduced from 1800 kg t... 0 kg.

In view of these s...ies, it seems reasonable to conclude that the risk of using a small car is double that of using a large car. Roughly, we may take them to be 1.4 and 0.7 times the averages listed in Table 3, respectively. The LLE is about (1.4 × 207 =) 290 d with small cars and (0.7 × 207 =) 145 d with large cars. Within the uncertainty of these estimates, it is reasonable to esumate that using small cars throughout life reduces life expectancy by 70 d, and using large cars increases life expectancy by 70 d.

PASSIVE SMOKING

One of the largest risks in our society is cigarette smoking, which was treated in some detail in CR. In recent years, attention has shifted to exposure of non-smokers to tobacco smoke, often referred to as "passive smoking" or "involuntary smoking." The principal method of studying this is by investigating the incidence of lung cancer, which is highly specific as an effect of tobacco smoke, among non-smoking spouses of regular smokers. A recent review (Surgeon General 1986) includes 10 studies of this type and gives the relative risks for non-smoking spouses of smokers vs. non-smokers.

None of the 10 studies, by itself, gives convincing evidence that passive smoking is harmful; in fact, two of them give a negative result (relative risk < 1.0) implying that passive smoking is beneficial. However, these two have comparatively low power, whereas some of the other

Table 6. Ratios of mortality rates for various diseases for unmarried to married white men and women, 1959–1961 (NCHS 1970). LLE are the loss of life expectancy in days for the total population due to that cause and ... used to calculate the effect of being unmarried, as explained in the text.

Cause of death	Men LLE (d)	Single	Widowed	Divorced	Women LLE (d)	Single	Widowed	Divorced
All causes	—	1.48	1.54	2.23	—	1.30	1.45	1.44
Tuberculosis	—	3.82	2.17	6.67	—	2.37	1.43	2.45
Cancer-digestuve	230	1.25	1.26	1.55	260	1.15	1.23	1.16
-Respiratory	350	1.16	1.26	2.13	200	1.04	1.18	1.53
-Breast	—	—	—	—	180	1.46	1.11	1.14
-Genital organs	100	0.95	1.23	1.37	100	1.14	1.18	1.65
Leukemia	35	1.19	1.08	1.21	35	1.06	1.10	1.07
Diabetes	70	1.46	1.41	1.92	120	0.66	1.11	0.90
Stroke	390	1.37	1.50	1.81	630	1.28	1.47	1.35
Heart-artery disease	2,300	1.32	1.46	1.77	1,970	1.26	1.48	1.30
Cirrhosis of liver	130	2.57	2.42	6.22	85	0.84	1.31	2.64
Motor vehicle accidents	360	1.51	2.27	4.20	150	1.03	1.10	2.28
All other accidents	310	1.51	2.27	4.20	150	1.72	1.84	2.37
Suicide	131	1.53	2.39	4.08	62	1.16	1.66	3.19
Homicide	136	1.03	2.69	7.22	43	0.51	1.28	4.51

eight have rather high power. When the data from all studies are combined, a reasonably clear picture emerges with a relative risk of about 1.5 or more.

There is evidence that this risk estimate may be too high. A relative risk of 1.5 corresponds to the risk of smoking about three cigarettes per day, but the exposure to tobacco smoke from passive smoking is only equivalent to smoking 0.5–1.0 cigarette per day. Among those exposed to passive smoking, levels of continine, often taken as a biological marker for effects of tobacco smoke, correspond to what is expected from smoking about 0.25 cigarettes per day.

However, there is evidence that the risk may be higher. A relative risk of 1.5 for lung cancer in a non-smoker gives an LLE of 50 d. For regular smokers, the increased risk of lung cancer represents only 13% of the total excess risk from all causes due to smoking. (The principal excess risk is from cardiovascular diseases.) One could perhaps justify dividing the above 50 d by 0.13, giving a total LLE = 380 d, or about 1 y. Several studies of effects of passive smoking on incidence of cardiovascular disease have been reported and direct evidence has been reported for damage to the cardiovascular systems of non-smokers by association with smokers (Fackelmann 1990), but quantitative esumates are difficult to make at present. When all things are considered, the LLE = 50 d may be a reasonable first estimate.

In CR, the LLE from cigarette smoking was given as 6.8 y from one to two packs per day and 8.6 y from over two packs per day for males, and 3.5 y from one to two packs per day for females. The latest results (Lew and Garfinkel 1987) from one or more packs per day are 6.6 y for males and 3.9 y for females. For people in good health at age 35, the LLE are 7.1 y for males and 4.2 y for females.

AIR POLLUTION

Dozens of studies of the excess mortality due to air pollution have been reported. They generally deal with "cross sectional" studies in which correlations are reported between air pollution and mortality in a number of cities, or "time series" studies in which the time variation of air pollution and mortality rates in a particular city are correlated. Over a period of several years, there has been a concerted effort by a group from the Harvard University Energy and Environmental Policy Center to evaluate and analyze the data from all of these studies. Their "findings are consistent with, and seem to support, the suggestion that the mortality effects of urban air pollution can be 6% or more of the total deaths" (Ozkaynak and Spengler 1985). From private discussions with the authors, we discern that their best estimates are now in the range of 2–5%; a reasonable estimate would be 3%. If the U.S. population were in age equilibrium, there would be 3.2 million deaths per year; hence air pollution causes about 100,000 deaths per year. Since the average victim probably loses about 7 y of life expectancy, from eqn (9) LLE = (0.03 × 7 =) 0.21 y or 77 d. Since coal-burning power plants are responsible for about 30% of all air pollution, they probably cause approximately 30,000 deaths per year. This is considerably higher than the estimate of 10,000 deaths per year used in CR. It would raise the LLE to 23 d for the average American.

OTHER ENVIRONMENTAL POLLUTANTS: CANCER RISKS

An EPA study (EPA 1987) utilized numerous scientists with vast experience in assessing risks in their various fields of specialization to rank various categories of environmental problems according to the relative importance of the risks they present. These rankings consider the numbers of people exposed and the degree to which they are exposed. Risk estimates are based on the few agents on which adequate information is available, but the rankings include estimated effects of other agents that have yet to be identified or to have their health impact quantified. Clearly this exercise involved a great deal of subjective consideration, and the results can hardly be regarded as scientific conclusions. However, for many purposes, they are the results being sought, and since there is not enough scientific information available for deriving them, this is probably the best available approach.

EPA estimates are given in terms of deaths per year. Since these deal with exposures accumulated throughout life, we assume that these numbers refer to a life-table age distribution for the present U.S. population. The LLE are then calculated from eqn (7).

The EPA study treats cancer risks and non-cancer risks separately. We describe their respective results in the present and following sections. For cancer, we assume that each victim loses an average of 18 y of life expectancy; i.e., p = 18 y in eqn (7).

Cancer risks due to inhalation or ingestion of various chemicals, as judged by the EPA's panel of experts, are listed in the EPA study (1987) and were used in their evaluations. They ranked various problem areas in order of their importance to the health of the population. Their rankings were as follows (number of deaths per year in parentheses):

1 (tie). Indoor Rn (14,000)—LLE = 30 d (see separate section below).

1 (tie). Worker exposure to chemicals (14,000)—LLE = 30 d because it is tied with Rn. Effects are estimated from formaldehyde (100), methylene chloride (90), asbestos (50), and tetrachloroethylene (10), plus the fact that there are 20,000 other chemicals that may give comparable risks (deaths per year in parentheses).

3. Pesticide residues on food (6,000)—LLE = 12 d. This result is derived from evidence on seven substances plus an estimate that there are about 200 other chemicals with comparable risks.

4 (tie). Indoor air pollution (3500–6500)—LLE = 10 d. The EPA summary gives "3500 to 6500" deaths per year, but the discussion gives 5,000 y^{-1} from tobacco smoke (in home and outside home contributing equally)

plus 1240 from six organic pollutants (benzene, paradichlorobenzene, chloroform, carbon tetrachloride, tetrachloroethylene, trichloroethylene) plus a few hundred from miscellaneous organic chemicals including formaldehyde from insulation, chlordane used to control termites, carcinogens produced by molds and fungi, asbestos, etc.

4(tie). Consumer product use (5000)—LLE = 10 d. The estimated deaths per year (in parentheses) from the analysis are: formaldehyde (50), methylene chloride (30), para-dichlorobenzene (30), and asbestos (5); there are 10,000 other chemicals that may contribute.

6. Carcinogens in air pollution (2000)—LLE $\approx$ 4 d. The principal contributors and the deaths per year (in parentheses) are: products of incomplete combustion (610) (e.g., benzo-a-pyrene), formaldehyde (435), waste treatment facility emissions (240), 1,3-butadiene (223), benzene (90), asbestos (82), gasoline vapors (77), chromium (75), carbon tetrachloride (69), ethylene oxide (58), and methyl chloride (35).

7. Stratospheric ozone depletion. It is estimated that this will cause 10,000 deaths per year (LLE of 22 d) by the year 2100 due to increased ultraviolet radiation.

8. Inactive hazardous waste sites (1000+)—LLE = 2.5 d as a national average. This includes the 35 "superfund" sites: 17.5 deaths per year, LLE = 4 d to the 30,000 people exposed. For the nationwide problem, 59% are due to vinyl chloride, 25% are due to trichloroethylene, and 14% are due to arsenic.

9. Drinking water contaminants (400–1000)—LLE = 1.3 d as a national average. Over half of the problem is due to trihalomethanes from chlorination, and over 20% is due to Rn. Less than 10% of the deaths (numbers per year in parentheses) are due to heptachlor (42), vinyl chloride (40), Ra (30), carbon tetrachloride (12), and ethylene dibromide (8).

10, 11. Not applicable.

12. Consumer use of pesticides (150)—LLE = 8 h.

13. Active hazardous waste sites (<100)—LLE <5 h. The only quantitative estimates are landfills and impoundments (35), incinerators (5), waste oil (2), and burning in furnaces (0.3).

14. Non-hazardous industrial waste sites with release through ground water (60)—LLE = 3 h. Principal contributors are arsenic, chloroform, benzene, and tetrachloroethane from the steel, paper, and organic chemical industries. The EPA panel did not give a risk estimate, but our estimate is deduced from the ranking.

15. New toxic chemicals (60)—LLE = 3 h, deduced from the ranking.

16. Municipal landfills and incinerators (40)—LLE = 2 h.

17. Contaminated sludge (40)—LLE = 2 h. This is due to using this sludge as fertilizer, disposing of it in landfills, incinerating it, or dumping it in oceans and contaminating seafood.

18. Mining, smelting, and refining waste released through ground water (10–20)—LLE = 45 min.

19. Releases from gasoline storage tanks into ground water (<1)—LLE = 3 min.

20, 21. Miscellaneous releases into ground water (<1)—LLE = 3 min. The only quantitative estimate was of methylene chloride from septic systems.

The EPA panel considered several other pollution sources but concluded that none was as important.

OTHER ENVIRONMENTAL POLLUTANTS: NON-CANCER HEALTH RISKS

Another EPA panel considered non-cancer health risks. These are much more difficult to estimate quantitatively; therefore, few quantitative estimates are given. The basic conclusions are expressed by characterizing the risks of each class of pollutant as "high," "medium," "low," and "unranked." "Unranked" implies that they are negligible. It is stated that the difference in risk magnitude between successive categories (e.g., high and medium risk) is at least two orders of magnitude but that there is no significance to the order of listing within the category.

The high risk category includes air pollution components on which EPA maintains standards: Pb, CO, SO_2, particulates, acid aerosols, NO_x, and ozone. We have previously ascribed 100,000 deaths per year to these from the conclusions of the Harvard Study, but less elaborate studies have derived estimates of about 10,000 deaths per year. It was probably a number of this order that participants in the EPA study had in mind when they concluded that there are no large differences within the category. This implies about 10,000 deaths per year due to each of the other classes in the high risk category.

The medium risk category includes a few risks that are quantified: Rn, radiation other than Rn, and ultraviolet radiation due to ozone depletion. The first two are included as non-cancer risks because of their genetic and teratological effects, which can be quantified. They each are estimated to cause about 200 serious cases per year that might be interpreted as equivalent to about 100 deaths per year. The ultraviolet radiation is estimated to cause about 20,000 cataracts per year, which again might be interpreted as crudely equivalent to 100 deaths per year. (Cataracts are located in one eye and are almost always effectively removed by surgery.)

From these three classes that are classified as "medium" risk, we conclude that "medium" risk corresponds to roughly 100 deaths per year. Since the high risk category is stated to be more than two orders of magnitude more serious, we conclude that each element of the high risk category causes more than 10,000 deaths per year in the U.S. This is in agreement with our previous conclusion from the fact that air pollution due to major pollutants is included as a non-exceptional member of that category. We crudely estimate that an average victim dies 15 y prematurely. Using this in eqn (7) gives, roughly, LLE = 20 d. This LLE then applies crudely to each class in the high risk category. These are:

1. Hazardous air pollutants other than those regulated by EPA. Substances included in the analyses were benzene, carbon tetrachloride, chlorine, chromium, formaldehyde, and hydrogen sulfide, but these are thought to represent less than 3% of the substances in this class.

2. Indoor air pollutants other than Rn. Although six substances were considered, this class is dominated by environmental tobacco smoke.

3. Materials in drinking water. Those included were lead, pathogens, legionella, nitrates, and chlorine disinfectants, and these are estimated to cause 30–100% of the risk.

4. Pesticide residues on food. The estimates were based only on aldicarb, diazinon, and EPN, but these are believed to represent less than 3% of the problem.

5. Consumer product exposure. Substances considered were methylene chloride (believed to be most important), 2-ethoxyethanol, and formaldehyde, but these are estimated to cause only 3–10% of the risk from this class.

6. Worker exposure to chemicals. Substances included are those in consumer product exposure (point no. 5) plus perchloroethylene, but these are believed to represent less than 3% of the total effect.

Other classes in the high risk category apply to workers in specific industries, chemical workers, and pesticide applicators.

The medium risk category gives risks about 100 times smaller, corresponding to an LLE of about 0.2 d. In addition to the radiation and ultraviolet classes mentioned previously, these include the following: releases from sewage treatment systems: non-point sources of discharges to surface water (e.g., rainwater washing out fertilizer and pesticides, and air pollutants settling into water); municipal landfills, incinerators, impoundments, etc.; and industrial counterparts of these miscellaneous pesticide contamination.

The low risk category gives risks a hundred times smaller than these, corresponding to an LLE of a few minutes. These include industrial discharges into surface water, sludge from sewage treatment plants, hazardous waste sites (active or inactive), mining wastes, releases from petroleum storage tanks, and farm storage facilities.

PURPOSELY INGESTED SUBSTANCES

Crouch and Wilson (1982) have developed risk estimates for carcinogens occurring naturally in foods. We express them here as LLE:

- broiled meat (organics by pyrolysis)—0.13 kg d^{-1} (2 lb wk^{-1}) LLE = 3 h
- peanut butter (aflotoxin)—1 tbsp. (14 g) d^{-1} LLE = 1 d
- milk (aflotoxin)—0.45 L d^{-1} (1 pint d^{-1}) LLE = 0.9 d
- chlorinated water (chloriform), Miami, FL LLE = 0.5 d

In CR, it was estimated that alcohol abuse causes 56,000 deaths per year in the United States due to direct effects (cirrhosis of the liver, alcoholic psychosis), accidents, suicides, and homicides. A more recent estimate (Ravenholt 1984) raises this to 100,000. If we assume that each victim loses about 20 y of life expectancy, applying eqn (6) gives an LLE = 1 y for the average American. Abuse of other addictive substances, estimated in CR to cause 6,000 deaths per year, has been more recently estimated (Ravenholt 1984) to cause 35,000, corresponding to an LLE = 125 d averaged over the population.

Of course, risks are much higher to those involved in substance abuse. For example, insurance studies have shown that "alcoholics" have mortality rates 2.5–3 times that of the general population. Using these in eqn (5) gives LLEs = 12.8 and 15.5 y, respectively. If the increased mortality rate does not begin until age 25, these LLE are reduced to 10.8 and 12.9 y.

Oral contraceptives increase a woman's risk of heart attacks and strokes by an amount depending on age and smoking habits (RCGP 1977, 1981); using them from age 15–45 reduces life expectancy by 107 d for smokers and by 25 d for non-smokers according to calculations based on eqns (1) to (3). Evidence indicates that effects linger after use is discontinued. If they linger to age 55, these LLE are increased to 293 d for smokers and 70 d for non-smokers. These numbers are much larger than those given in CR, based on phlebitis.

During the 1950s, diethylstilbestrol (DES) was used to prevent miscarriages, but it was later found that daughters of DES users had an increased incidence of vaginal cancer, with a risk of 0.1% (Melnick et al. 1987). According to eqn (5), this corresponds to an LLE of about 4.7 d to the daughters.

According to death certificate information (NSC 1989), poisoning by drugs, medicaments, and biologicals

Table 7. LLE due to sports participation per year of participation. The second column gives the probability per year of being killed (Reif 1981), the third column gives an estimate of the average life expectancy lost by a victim, and the last column gives the product of these × 365, which is the LLE in days.

Sport	Probability of death	Years lost per victim	LLE (d)
Prof. boxing	1:2,200	50	8
Hang gliding	1:560	40	25
Football			
-high school	1:81,000	60	0.3
Football			
-college	1:33,000	55	0.6
Mountain climbing			
-dedicated	1:167	50	110
-all climbers	1:1,750	50	10
Mountain hiking	1:15,700	40	0.9
Parachuting	1:570	40	25
Sail planing	1:1,710	40	9
Scuba diving (amateur)	1:2,400	45	7
Skiing-racing	1:40,000	50	0.5
Snowmobiling	1:7,600	40	2

caused an average of 4,200 deaths per year in 1984–1986. If each victim lost an average of 20 y of life expectancy, this is an LLE of 10 d averaged over the U.S. population according to eqn (7). Of these deaths, 27% were from pain relievers (e.g., aspirin), 6.5% were from tranquilizers and psychotropic agents, 13% were from other drugs acting on the nervous system, 2% were from sedatives, and 1.5% were from antibiotics and anti-infectants.

SPORTS PARTICIPATION

The probability of being killed in a sports accident and the LLE per year of participation in that sport are listed in Table 7 (Reif 1981). The method of calculation is explained in the caption.

A jogging study conducted in Rhode Island (Thompson et al. 1982) showed that 7.4% of all men between ages 30–64 jog at least twice a week, and there were 12 deaths attributed to this exercise in 1975–1980. The risk per jogger was 1/7,620 per year. Assuming 35 y lost life expectancy per victim, according to eqn (9), this corresponds to an LLE of 1.7 d per year of jogging or 50 d from 30 y of participation. Jogging is usually viewed as a measure for preventing heart disease that has an LLE = 2,100 d, so if 30 y of jogging reduces the probability of heart disease by more than (50/2100 =) 2.4%, it is worth the risk.

GEOGRAPHY

In CR, it was shown that life expectancy varies by about 2.5 y depending on the region of the country where one resides, with the shortest life expectancies in the Southeast (SC, MS, GA, LA, AL in that order) and the longest in the Northern Plains (ND, MN, SD, NB, KS, IA, WI in that order). It was shown that there is little correlation with annual income and that it is reasonable, therefore, to consider geography as a risk factor with an LLE of plus or minus more than a year. A more recent study (NCHS 1980) of geographic patterns in the risk of dying between ages 35–74 calls this "the enigma of the southeast" and searches for explanations. The principal contributor is cardiovascular diseases, and the effect is larger in men than in women. The principal possible explanation considered is low levels of trace elements in the soil. Note that the quantities of naturally occurring trace elements are very much larger than the quantities of these elements released into the ground by technology.

That report considers other observed geographic variations in life expectancy. Areas with heavy mining have consistently high mortality rates for both males and females. It is speculated that mine products (e.g., coal dust) or mine wastes (e.g., sulfur) may cause health problems through air or water pollution, although cultural and socioeconomic factors are also considered. No data are presented that can be used to calculate LLE, but it is clearly a matter of a few years.

Areas at high elevation have substantially lower mortality rates than areas near sea level. The correlations are especially strong for cancer, but they are also significant for cardiovascular disease. Again no data are given for estimating LLE, but it is a few years. The report also considers population density and rural vs. urban effects and concludes that the difference is small, in agreement with CR.

MEDICAL CARE

Progress in medical technology is widely recognized as giving important life-saving effects, especially by reducing risks from cardiovascular disease. Between 1970 and 1980, mortality rates decreased by an average of 2% y^{-1}, increasing life expectancy by 3.1 y (Myers and Bayo 1985), with 70% of this due to progress in cardiovascular diseases. It is instructive to consider individual advances.

There were 332,000 coronary artery bypass operations performed in the U.S. in 1987, with many of them averting imminent death.[†] If we assume that each operation adds an average of 3 y to the life expectancy of the patient, applying eqn (6) indicates that this technology adds a half year (180 d) to our life expectancy (LLE = −180 d). These operations first became available in 1970 and were not applied widely until about 1980.

Coronary pacemakers first became available in 1960 and are now being implanted at a rate of about 500,000 per year.[‡] If we assume that they extend life expectancy of patients by an average of 2 y, application of eqn (6) indicates that they add 180 d to the life expectancy of the average American (LLE = −180 d).

There are about 9,000 kidney transplants each year in the United States.[§] Survival after 5 and 10 y is 75% and 55%, respectively (40% higher if the donor is a sibling, 40% lower if not). From these data we estimate the average added life expectancy from each transplant to be 12 y. According to eqn (6) these operations, which first became available in 1955, add 20 d to the life expectancy of the average American (LLE = −20 d).

About 110,000 Americans are receiving kidney dialysis, and the average duration of such treatment is about 5 y before death or reception of a kidney transplant. This implies that 5 y of life expectancy is added to 22,000 people averted each year. Application of eqn (6) indicates that kidney dialysis technology, which first became available in 1945, is adding 20 d to the life expectancy of the average American (LLE = −20 d).

Use of β blocker drugs following heart attacks is estimated to save 17,000 lives per year in the U.S. (Urquart and Heilmann 1984). If the average would-be victim gains

[†] Personal communication (1990), N. Haas, American Heart Association, Dallas, TX.

[‡] Personal communication (1990), S. Song, University of Southern California Cardiac Pacemaker Center, Los Angeles, CA.

[§] Personal communication (1990), D. Zobel, U.S. Renal Data System, Washington, DC.

5 y of life expectancy from its use, according to eqn (6) the average American gains 15 d of life expectancy (LLE = −15 d).

It is interesting that most people benefitting from coronary artery bypass operations, pacemakers, and β blocker drugs will still die of heart disease, so the effects of these technological advances are not apparent in mortality rates from that cause. This illustrates one of the advantages of using life expectancy as a measure in considering effects of risks.

Hadley (1982) estimates that a 10% increase in the number of physicians would result in an 0.8% decrease in mortality rates (p. 154) and, elsewhere in the same book (p. 10), estimates that a 10% increase in medical care would reduce mortality rates by 1.5%. Auster et al. (1969) estimate that a 1% increase in medical services would reduce mortality rates by 0.1%, two-thirds of Hadley's maximum estimate (assuming linearity). Converting to a risk viewpoint, we conclude that a 1% reduction in our medical services might increase mortality rates by about 0.1%, giving us an LLE = 5 d according to eqn (5).

However, a geographical correlation study (NCHS 1980) finds, surprisingly, a *positive* correlation between the number of physicians and mortality rates, although the correlation with number of general practitioners is negative. Robin (1984) takes a strong position that we have far too many physicians in the United States. He notes that U.S. and Canada have twice as many physicians per capita as Great Britain, but there is no difference in mortality rates from various diseases, including those treated by surgery. He also cites studies of physician strikes in Canada, Israel, and Southern California during each of which mortality rates declined, returning to their normal levels after the strikes were ended.

Another contrary view is the estimate by Sagan (1987) that hospital-acquired infections may cause up to 100,000 deaths per year. If we estimate that an average case causes the victim to lose 10 y of life expectancy, application of eqn (6) indicates that this corresponds to LLE = 0.5 y.

Ravenholt (1987) estimates that failure to detect and treat treatable cancers—especially cancers of the cervix, skin, colon, breast, prostate, and bladder—cause 75,000 avertable cancer deaths per year in the U.S. (15% of all cancer deaths). According to Table 2, this corresponds to an LLE = 187 d (0.5 y) due to inadequate cancer screening. He also estimated that 125,000 unnecessary deaths per year are caused by inadequate control of high blood pressure, which is 13% of all deaths from heart disease and stroke. According to Table 2, this corresponds to an LLE = 0.7 y. Adding these to the estimate from CR that improper use of drugs in medical treatment causes LLE = 0.25 y (confirmed by Ravenholt), the total effect of deaths preventable by good medical services with technology now available is LLE (= 0.5 + 0.7 + 0.25) = 1.45 y = 530 d. However, Robin (1984) questions the advantages of medical screening programs.

Acquired immune deficiency syndrome (AIDS) is a preventable disease caused, directly or indirectly, by promiscuous sexual practice. It is killing about 12,000 Americans per year, with each victim losing about 40 y of life expectancy. Averaged over the U.S. population, this gives an LLE = 55 d according to eqn (7).

If everyone would use all currently available technology to prolong life, including good dietary practice, proper exercise, proper sleep, and the best available medical care, Ravenholt (1987) estimates that all age-specific mortality rates would be cut in half; that, according to eqn (5), would increase life expectancy by 9.0 y. Thus, sub-optimal practices cause an LLE = 9.0 y. Cigarette smoking is the largest single contributor to this risk.

Medical progress extends life expectancy. As one quantitative example, it is estimated that the cancer cure rate has been improving about 1% per year (Cohen 1983). Since our LLE from cancer is 1247 d, progress in cancer treatment each year gives an LLE = −12 d.

EPIDEMICS

Historically, disease epidemics have been one of the most important human risks (Cornell 1979). "The Plague of Justinian" killed 100 million people in A.D. 500–650. The "black death," in 1347–1351, killed 75 million people in Europe (one person in four), wiping out 200,000 villages; it may have done even more damage in Asia, reducing the world population by as much as one-third. The influenza epidemic of 1918–1919 killed 25–50 million people (another estimate is 20 million, Weiss 1989), including 500,000 in the U.S. Rat-born diseases killed 12

Table 8. Average number of deaths per year in the United States and LLE from some natural hazards. *P* is the average loss of life expectancy (in years) per victim assumed in calculating LLE from eqn (6).

Hazard[a]	Deaths per year	*P*	LLE (d)
Hurricanes	41	35	0.3
Tornadoes	124	35	0.8
Excessive heat	236	15	0.6
-NSC	270	15	0.7
Excessive cold	366	15	1.0
-NSC	770	15	2.1
Lightning	141	45	1.1
-NSC	85	45	0.7
Floods	62	35	0.4
Earthquakes	28	35	0.2
Tsunami	24	35	0.15
Weather-related transportation accidents	288	35	1.8
Venomous plants, animals	60	45	0.5
-Snakes, lizards, spiders	10	45	0.08
-Hornets, wasps, bees	44	45	0.4
Dog bites	15	45	0.12
Storms and floods	140	35	0.9
Earthquakes and volcanoes	20	35	0.13

[a] Above line: information from Cornell (1979). Below line and designated "NSC": information from National Safety Council (1989).

million people in India in 1898–1923. In the 16th century, small pox killed several million—up to half of the native Indian population encountered by the Spanish Conquistadors. In 1530–1545 measles killed an additional 1.5–2 million. Typhus killed 3 million people in Eastern Europe in 1914–1915.

Many dozens of more localized epidemics have been recorded. For example, 280,000 Crusaders, 90% of those returning from the Holy Land, died of an unknown disease in 1098–1101, and the "Great Plague" killed 70,000 in London during the summer of 1665. A listing of epidemics in China (McNeill 1976) includes an average of about eight per century from the first to the 14th, and about 40 per century from the 16th to the 19th. In some of the earlier ones, half or more of the local population died.

Medical science in advanced nations is now powerful enough to have prevented most of these epidemics (although perhaps not the influenza epidemic of 1918–1919). However, the AIDS virus has now infected 5–10 million people with no end in sight, and in 1983, a poultry virus in Pennsylvania killed 17 million chickens in 6 mo (Weiss 1989). Such epidemics can arise from a mutation in a virus, and mutations in viruses occur very frequently (one in 10,000 replications). There have been numerous localized outbreaks from this source. Rift Valley fever, a mutation of a virus normally found in sheep, killed thousands of Egyptians in 1977. The Ebola virus killed 500 in Zaire and Sudan in 1976. Epidemics of hemorrhagic diseases have occurred recently in Bolivia and Argentina, and there was a similar outbreak in Nigeria.

None of these diseases has spread worldwide except for AIDS which, fortunately, is not spread through highly efficient channels such as coughing and ingestion of food, but there is no reason why a virus could not develop that would spread in those ways. Modern technology has provided new and highly efficient transport modes for viruses, such as blood transfusions and greatly expanded human travel. Many of the chemicals introduced by our technology are mutagens and could, therefore, induce such an unfortunate mutation.

NATURAL HAZARDS

The number of deaths per year from various natural hazards and their associated LLE are listed in Table 8

Table 9. Life expectancy in 1960 in various sections of Chicago classified according to socioeconomic status: 1 = low, 5 = high (Kitagawa and Hauser 1973).

Socioeconomic status	White males	White females	Other males	Other females
1	60.0	67.7	56.7	62.5
2	64.6	71.2	59.9	65.1
3	66.5	72.8	65.1	68.1
4	67.9	73.8	—	—
5	67.4	73.6	—	—
Suburbs	69.0	74.6	66.1	72.3

Table 10. Ratio of crude mortality rates in poverty and non-poverty areas of various U.S. cities (NCHS 1975).

City	Whites	Others
Atlanta	1.78	1.92
Baltimore	1.45	1.74
Buffalo	1.90	1.89
Chicago	1.44	1.70
Cincinnati	1.72	1.60
Cleveland	1.48	1.56
Dallas	1.72	1.69
Denver	1.60	2.40
Indianapolis	2.39	1.75
Memphis	2.08	1.20
Minneapolis	1.31	1.18
New York	0.85	1.33
Philadelphia	1.41	1.60
Pittsburgh	1.55	1.42
San Diego	1.50	2.28
San Francisco	1.74	1.75
Seattle	1.96	1.92
Washington	1.84	1.55
Ave. =	1.71	1.70

(Cornell 1979; NSC 1989). We see that the LLE are generally about 1 d or less, and added together, they are less than a week. Bee stings give a risk of 1.8×10^{-7} y^{-1} (Rubenstein 1982) which, according to eqn (4), gives an LLE = 0.2 d = 4.8 h. The added risk of death at all ages from being struck by lightening is estimated by Urquhart and Heilmann (1984) to be 5.3×10^{-7} y^{-1}, corresponding to an LLE = 0.6 d according to eqn (4). Estimates in Table 8 are 1.1 and 0.7 d.

While the data in Table 8 give the impression that natural hazards are not important sources of risk, individual incidents may cause heavy loss of life. A 1970 hurricane in Bangladesh killed a million people, and there have been four others in India and Vietnam that killed over 100,000. The most disastrous U.S. hurricanes killed 6,000 in Galveston in 1900, 2,000 in Florida in 1928, and 1800 in New Orleans in 1893. U.S. tornadoes killed 689 in 1925 (Midwest), 600 in 1884 (South), 300–400 in 1896 (St. Louis), 419 in 1936 (MS and GA), 343 in 1942 (MO to AL), 315 in 1974 (Midwest), 300 in 1840 (MS), 272 in 1965 (Midwest), 268 in 1932 (AL), etc.

On several occasions, floods have killed hundreds of thousands of people in China, but the most disastrous U.S. floods killed 500–700 in 1913 (Ohio River), 313 in 1927 (Mississippi River), 250 in 1937 (Mississippi River), and 190 in 1955 (Connecticut River). The famous Johnstown flood that killed 2,200 in 1889 was due to collapse of a dam; this is not usually classed as a natural disaster, but weather contributed to the dam's collapse. Other floods caused by heavy rainfall-induced dam collapse drowned 450–700 people near Los Angeles in 1928, over 200 near Rapid City, SD in 1972, 144 in Williamsburg, MA in 1874, and 125 in Buffalo Creek, WV in 1972. There are two dams in California whose collapse could cause 200,000 deaths (Okrent et al. 1974).

A few Chinese earthquakes have caused over a half million deaths, and many tens of thousands have died in

earthquakes in Japan, Italy, Caucasia, Greece, Portugal, Peru, Ecuador, Chile, Colombia, and Venezuela. The most disastrous U.S. earthquake (San Francisco, 1906) killed about 700. Tsunamis have killed tens of thousands in Indonesia, Japan, and Portugal, but the highest U.S. death tolls were 173 (1946) and 61 (1960), both in Hawaii.

While disasters resulting from natural causes have been minimal in the U.S., they are always a possibility. It is estimated that an earthquake in Los Angeles (~7.5 on a Richter scale) could cause 20,000 to 200,000 deaths; one elaborate study (NOAA 1973) estimated 40,000 deaths.

SOCIOECONOMIC FACTORS

Included in CR was a great deal of evidence that higher socioeconomic status increases life expectancy. Much more data pertaining to this are now available. Kitagawa and Hauser (1973) classified various sections of the Chicago area according to socioeconomic class and determined the average age at death, which is closely correlated with life expectancy. Results are shown in Table 9. We see that life expectancy is longer for the highest than for the lowest socioeconomic status citizens by 9 y for white males, 7 y for white females, and 10 y for nonwhites. These are the LLE from being poor rather than well-to-do. A U.S. Public Health Service study (NCHS 1975) for 1969–1971 classified census tracts as "poverty areas" if over 20% of its population had incomes inadequate to meet basic nutritional needs. For example, for a family of four this was U.S. $3743 per year. It then compared mortality ratios in poverty areas and non-poverty areas of 19 large cities. The ratio of crude mortality rates between poverty and non-poverty areas is listed in Table 10. We see that, with one exception, this ratio is substantially greater than 1.0 for both whites and non-whites in all cities. The average ratio is 1.70. According to eqn (5), this corresponds to an LLE = 6.9 y.

However, before making inferences about life expectancy, crude mortality rates must be age-adjusted because areas with younger inhabitants naturally have lower crude mortality rates. Data for age-adjustment are not available, but another report (U.S. Bureau of the Census 1973) finds that populations are *younger* in poverty areas. For the 50 largest cities, the median age is 29.3 y for whites and 23.4 for non-whites in poverty areas vs. 33.0 and 25.6 y, respectively, in non-poverty areas. This means that ratios of age-adjusted mortality rates are substantially higher than those listed in Table 10. The average ratio is probably more than 2.0 which, from eqn (5), gives an LLE > 9.0 y. Note that Chicago, which had a 7–10 y difference in 1960 according to Table 9, has a *below* average ratio in Table 10. Therefore, it seems reasonable to conclude that in most large American cities, poverty causes an LLE of about 10 y. This is substantially larger than the LLE of 4 y estimated in CR from data on occupational categories.

The British Registrar-General's report (Registrar-General 1978) classifies people by occupation into six groups (I, II, III N, III M, IV, V) according to "social class"; class I, which consists of professional people, is the highest, while class V is the lowest, consisting of unskilled laborers. Differences from the national average mortality rate and life expectancy are listed in Table 11, with the method of calculation explained in the caption. From the last column, we see that professional men live 2.6 y longer than average while unskilled laborers live 4.6 y less than average, a total difference of 7.2 y.

The standardized mortality ratios for various causes of death for each social class are listed in Table 12. We see that there is a strong correlation for a very wide variety of diseases, and it is even stronger for accidents and homicide.

The Canadian Ministry of Health and Welfare carried out a study (Wigle and Mao 1980) of life expectancy vs. average income in 2,228 census tracts in 21 metropolitan areas based on statistics for 1971. Census tracts were classified by income level as 1, 2, 3, 4, 5, corresponding to quintiles in the distribution of incomes in Canada. The median household income (in 1971) varied from less than $7500 in level 5 to over $11,000 in level 1. The average life expectancy (in years) for levels 1 and 5 for males was 72.5 and 66.3, and for females was 77.5 and 74.6, respectively. We see that the difference in life expectancy between class 1 and class 5—the top and bottom 20% by income level—is 6.2 y for males and 2.9 y for females. The ratios of mortality rates for income level 5 to income level 1 were the following: heart disease and stroke—1.32 (M), 1.15 (F); cancer—1.34 (M), 1.07 (F); accidents, poison, and violence—1.88 (M), 1.46 (F). Of the 33 ratios of this type (17 diseases × two sexes), only two were less than 1.0, and one of these was 0.98. This is in agreement with conclusions from the U.S. (CR) and British (Table 12) data that poverty increases the risk of nearly every disease.

Table 11. Mortality for ages 15–64 by social class in England and Wales in 1970-1972 (Registrar-General 1978). The second column gives the difference in mortality rate from the national average which was 597×10^{-5} y^{-1}. The next two columns give the difference in life expectancy from the national average calculated from the second column assuming (1) the proportional difference in mortality rate stays the same after age 64, and (2) the difference in mortality rate becomes zero after age 64. The next column shows the average of the results of these assumptions that we take to be the best estimate of the actual situation, and hence the actual LLE. The last column converts this into years.

		Difference in life expectancy (d)			
Difference in class mortality rate		Same after 65	Zero after 65	Mean	Years
I	-135×10^{-5}	−1240	−680	−960	−2.6
II	−111	−1020	−560	−790	−2.2
III N	−6	−55	−30	−42	−0.1
III M	+36	330	+180	+260	+0.7
IV	+84	770	420	+600	+1.6
V	+235	2160	+1180	+1670	+4.6

Table 12. Standardized mortality ratios (SMR) for various causes of death in England and Wales vs. social class (Registrar-General 1978). SMR is 100 times the ratio of mortality rate to the national average mortality rate for that disease.

	Social class					
	I	II	III N	III M	IV	V
Tuberculosis	61	63	88	86	126	197
Syphilis	46	44	71	94	125	272
Malignant neoplasms	75	80	91	113	116	131
-Stomach	50	66	79	118	125	147
-Trachea, bronchus, lung	53	68	84	118	123	143
Circulatory system	86	89	110	106	110	118
-Hypertensive	71	85	104	104	112	141
-Cerebrovascular	80	86	98	106	111	136
Respiratory system	37	53	80	106	123	187
-Influenza	38	61	79	100	115	189
-Pneumonia	41	53	78	92	115	195
-Bronchitis, emphysema	36	51	82	113	128	188
Digestive system	83	91	97	92	109	152
-Ulcers	49	61	88	102	116	180
Genito-urinary system	63	81	102	98	111	152
Nephritis-nephrosis	58	83	103	103	109	152
Accidents-poisoning-violence	78	78	83	94	122	197
-Motor vehicle	77	83	89	105	121	174
-Motor cyclist	46	43	61	119	183	192
-Pedestrian	40	37	54	92	120	342
-Poisoning by gases	66	82	81	90	119	184
-Falls	49	47	46	103	131	265
Homicide	34	80	47	87	123	339

A study of different sections of Montreal (Wilkins 1986) gave the results listed in Table 13. The ordering is by decreasing average household income in 1970, but it is evident that essentially the same order would apply if based on the percent of heads of households with a professional occupation, with less than 9 y of education, or with a university education. We see that the difference in life expectancy between the highest and lowest socioeconomic level sections is 10.8 y for men and 7.3 y for women.

In census data for Finland, all men except farmers are assigned to social group I (highest), II, III, IV (lowest) based on occupations, or former occupations for retirees and the unemployed. The percentage of the total population in each and their life expectancies in 1969–1972 were (Näyhä 1977): I (7%), 67.5 y; II (23%), 65.5 y; III (31%), 66.1 y; IV (12%), 60.3 y, a 7.2 y difference.

A study based on data from France (Ledermann 1960) in the 1950s concludes that professionals and managers lived 13 y longer than miners and unskilled laborers.

In summary, data from U.S., Britain, Canada, Finland, and France indicate that, in technologically advanced nations, the LLE due to poverty is in the range 7–10 y. There can be no question that wealth brings health and poverty kills.

One of the reasons for this, to be discussed below, is the negative correlation between education (closely correlated with wealth and social class) and cigarette smoking. There are analogous correlations for alcoholism (Kissin and Begleiter 1976) and obesity (Silverstone 1969), both of which have serious health effects. Men in the highest social position included only 9% with alcohol problems, whereas among those in the lowest social position, 26% had alcohol problems. A study of 20–39 y olds in London found that in the upper social class, 17% of the men and 27% of the women were obese, whereas in the lower social class, 30% of the men and 70% of the women were obese.

Within technologically advanced nations, there are effective programs to provide poor people with reasonable medical care and an adequate diet, but such programs are much fewer and less effective on an international basis. One would, therefore, expect variations of life expectancy with socioeconomic level to be much larger. A test of this concept is shown in Fig. 1, which is a plot of life expectancy vs. gross national product (GNP) for the various nations of the world (World Resources Institute 1987). The very strong correlation is immediately obvious, not only for the world as a whole but within each continent individually.

The reason for the strong correlation between life expectancy and GNP shown in Fig. 1 has been explored in some detail by the World Bank to determine the most productive areas for investment (World Bank 1980). In correlation studies of life expectancy vs. various other factors, it was found that literacy was the most important factor and that distribution of income was more important than average income. The daily calorie intake and physicians per capita were of some, but lesser, importance. The World Bank concluded that *education* is the key factor affecting life expectancy, largely because the mother's education affects the health of her children: adults seem to adjust much more readily to their diet and environment, and even *their* health is heavily influenced by their childhood experience. The study found that each additional

Table 13. Socioeconomic factors and life expectancy in various districts of Montreal (Wilkins 1986).

District	Household income ($)	Occupational professional (%)	Education		Life expectancy	
			Percent <9 y	Percent University-educated	Male	Female
NDG-East/Westmount	14,500	18.9	13.8	43.5	72.2	78.2
NDG-West/Mtl-West	10,300	13.0	20.9	32.0	70.7	78.3
Metro	10,400	12.7	15.1	49.6	67.8	75.9
St. Louis/Mile End	6,600	2.6	53.0	14.6	66.4	75.2
St. Henry/L. Bergundy	6,500	2.6	53.9	6.7	61.4	

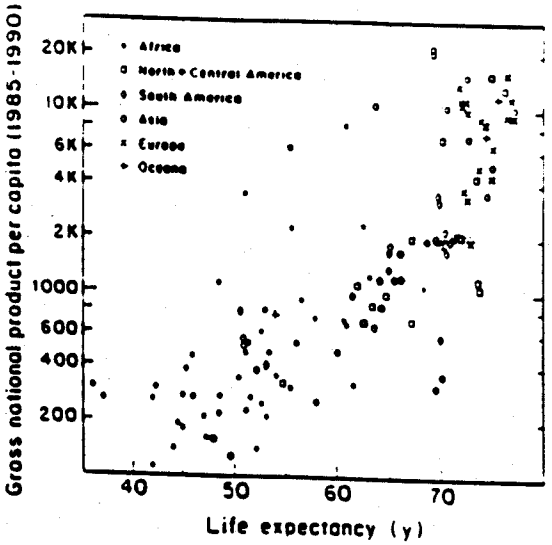

Fig. 1. Correlation between gross national product per capita and life expectancy in all countries for which data are available.

year of maternal education reduces child mortality by 0.9%. Thus, if the national life expectancy is 50 y, for each year lost from a mother's education, the child has an LLE of 0.45 y.

An interesting study corroborating this conclusion involved correlating infant mortality (IM) with female literacy (FL) in the various states of India (United Nations 1983). The state with lowest IM (Kerala) had the highest FL, although its per capita domestic product (DP) was moderate. Punjab and Haryana had the highest DP, 1.69 and 1.51 times higher than Kerala, but their FL was only 53% and 34% of Kerala's, and their IM was 2.0 and 2.2 times higher. For the 14 states, there was little correlation of IM with DP but a strong negative correlation of IM with FL.

Education is also an important factor influencing life expectancy in advanced nations. Direct evidence on this was given in CR, and further evidence has been cited by Sagan (1987). One study showed a strong negative correlation between college education and smoking (Covey and Wynder 1981). For physicians, dentists, lawyers, engineers, teachers, and clergymen—over 95% college educated—only about 17% were cigarette smokers, but for painters, carpenters, and taxi-, truck-, and bus drivers—only 6% of them college educated—46% were cigarette smokers. Data from other occupations fall into line in this smoking-education correlation.

It is interesting to understand the direct causes of early death in underdeveloped countries. Data are available for Ghana (GHAPT 1981) that can be used to estimate sources of LLE. Infant mortality there is about 10%, reducing life expectancy by about 5.7 y. In advanced nations it is about 1%, which reduces life expectancy by 0.7 y and gives Ghana an added LLE = 5 y from that source.

A life table is given in the reference, allowing us to calculate LLE from eqns (1), (2), and (3). The most important killer disease is pneumonia, which is 40% fatal among children giving LLE = 1.3 y and 10% fatal for adults, adding LLE = 0.6 y for a total LLE of 1.9 y (vs. 0.28 y in the U.S.). Measles have been virtually eliminated in advanced nations by immunization (uncommon in Ghana) so that disease gives an LLE = 1.3 y. The worst tropical disease is malaria with an LLE = 1.2 y. Actually, the worst effects of malaria are the debilitation it causes throughout life. Severe malnutrition (i.e., starvation) kills many young children, giving an LLE = 1.2 y (vs. 0.01 y in the U.S.). Sickle cell disease, which is an indirect genetic effect of malaria, kills many infants leading to an LLE of 1.2 y. Diarrhea, caused by problems with food, kills about 2% of all children before they reach age 5, contributing LLE = 1.0 y. (Most of these could be saved easily and cheaply with oral rehydration therapy). Tetanus and pertussis (whooping cough), both eliminated in advanced nations by immunization, kill infants and young children giving LLE = 0.5 y and 0.3 y, respectively. Schistosomiasis, a tropical disease, is 4% fatal to young children, giving LLE = 0.4 y.

Tuberculosis is still an important killer in the middle ages, giving an LLE = 0.5 y (vs. 0.01 y in U.S.). Other middle-age killers are cirrhosis with LLE = 0.5 y, intestinal obstructions with LLE = 0.35 y, complications of pregnancy with LLE = 0.65 y for females, and typhoid with LLE = 0.3 y. Even leprosy is still a factor, with LLE = 0.13 y.

The fact that few people live to old age greatly reduces the importance of adult cancer, which has an LLE of only 0.25 y vs. 3.4 y in the United States. The same is true of heart disease with LLE = 0.35 y vs. 4.4 y in the U.S., although here it is believed that dietary factors also contribute to the difference.

Of course, the fact that total life expectancy in Ghana is 20 y shorter than in the U.S. substantially reduces all LLE for Ghana.

RADON IN HOMES AND OTHER RADIATION

The National Academy of Sciences Committee (NAS 1988) estimates that, if the average Rn levels in U.S. homes is 37 Bq m^{-3} (1 pCi L^{-1}), 14,000 Americans per year die from Rn-induced lung cancer. If the U.S. population were in age equilibrium, there would be 3.2 million deaths per year in the U.S., which means that (14,000/3.2×10^6 =) 0.44% of all deaths are due to Rn. Since an average victim loses about 18 y of life expectancy, application of eqn (8) gives an LLE = 29 d for the average American. Many millions of American homes have a Rn level in excess of 450 Bq m^{-3} (12 pCi L^{-1}), which means they have LLE of a year or more from Rn.

The radiation risks quoted in CR require updating in view of the new dose estimates for the Japanese A-bomb survivors. Since most radiation exposures of interest are at low dose rates, there is a problem in choosing the "dose rate effectiveness factor" (DREF). The BEIR V Report (NAS 1990) does not recommend a value. NCRP Report No. 64 (1980) and the 1988 United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR 1988) recommend a DREF "between 2 and 10." The National Radiological Protection Board (1988)

recommends a DREF = 3 for all but breast cancer, for which DREF = 2. BEIR III (NAS 1980) includes an implicit DREF that varies with dose.

To obtain a DREF, a questionnaire was sent to all members of the Committees that authored BEIR III, BEIR V, NCRP No. 64, and UNSCEAR 1988, asking for personal best estimates of DREF. Geometric means of responses to each question are used, after converting all estimates greater than 10 (some were much higher) to a DREF = 10. Twelve responses offering numerical estimates were received. The mean DREF for these were:

- 1 mSv (100 mrem) y^{-1} for 70 y—3.4
- 0.01 mSv (1 mrem) y^{-1} for 70 y—4.2
- 13 mSv (1.3 rem) y^{-1} for 50 y—2.6
- single dose of 0.01 mSv (1 mrem)—3.9
- single dose of 10 mSv (1 rem)—2.7
- single dose of 100 mSv (10 rem)—1.9

Based on these results, we take DREF = 3 for exposures of 50 mSv (5 rem) y^{-1} or less, in agreement with the NRPB esumate. This leads to an age-averaged mortality risk esumate from BEIR V of 0.026/man-Sv (260×10^{-6}/man-rem). This cannot be used to calculate LLE by application of eqn (4) since death is delayed for many years after exposure.

The LLEs obtained from BEIR V, with application of DREF = 3, are:

- continuous exposure to 1 mSv (100 mrem) y^{-1} throughout life—LLE = 9.9 d;
- continuous exposure to 10 mSv (1 rem) y^{-1} from age 18 to 65—LLE = 51 d;
- single exposure to 10 mSv (1 rem), age-averaged—LLE = 1.5 d;
- single exposure to 0.01 mSv (1 mrem), age-averaged—LLE = 2.1 min.

According to Table 1–3 in BEIR V, average exposure from natural radiation, excluding Rn, is 0.94 mSv (94 mrem) y^{-1} which gives LLE = (0.94×9.9 =) 9.3 d. Medical radiation gives an average exposure of 0.63 mSv (63 mrem) y^{-1}, which causes LLE = 6.2 d. Average exposures to workers in the nuclear industry are now about 4.5 mSv (450 mrem) y^{-1} which (if experienced from age 18–65) gives LLE = 23 d.

ENERGY CONSERVATION

It is widely believed that the pollution caused by energy generation is so harmful that conserving energy would be a substantial benefit to our health, but energy conservation measures have their own risks that we can estimate by use of Tables 2 and 3, and the previous section.

Close to half of all American houses have been weatherized to save fuel, principally by weather stripping, caulking around windows, and closing gaps below doors: this increases the Rn level by about 10% (Cohen 1988), causing an average LLE = 3 d. The U.S. Department of Energy recommends, as a long-term goal, that air change rates in houses be cut in half. This would double the Rn concentration and give the public an average L = 29 d.

Another energy conservation measure is usi smaller cars, mandated by government directives. If t were equivalent to everyone shifting from mid-size small cars (as defined in the early 1970s), it would g the American public an LLE = 70 d. The actual shift h been by about half that much, giving us an LLE = 35

Another energy conservation measure is to redu lighting. Motor vehicle accidents are 2.5 times more lik to occur at night than in daylight (U.S. DOT 1981) seems reasonable to expect substantial reductions in str and highway lighting to increase traffic deaths by 5%. T gives an LLE (= 0.05×207) = 10 d.

Most homicides occur in the dark. Reduced light may increase their number by 5%, giving LLE = 4.5 d may also increase the frequency of falls by 5% giving L = 1.4 d. Cold (from conserving fuel) and darkness (fr reduced lighting) aggravate depression and might well crease the suicide rate by 5%, giving LLE = 6 d.

Bicycling is often employed as an energy conser tion measure, but it is much more dangerous per kilome than automobile driving. If bicycling doubles, the r spread over the entire population is LLE = 6 d. [A rec study by Thompson et al. (1990) indicates that this co be reduced to 0.8 d if use of helmets were to beco universal.]

The total risk of our present energy conservation fort probably approaches LLE = 50 d, whereas the ri of all of our energy generation were estimated in CR LLE = 24 d (increased to perhaps 34 d by the increa estimates on air pollution above). Thus, conservatio the *most dangerous* energy strategy. If we desist fr building power plants and developing energy sources the name of energy conservation, this could very ea discourage expansion of industry and thereby cause creased unemployment.

Myers and Werner (1987) estimate from histori analysis that each kilowatt (kW) per capita of energy co sumption adds 30 d of life expectancy in advanced cou tries and 220 d in less advanced countries. According this, each time a 1-million kW electric power plant, wh consumes 3 million kW of energy, is not built, aver life expectancy in the United States is reduced from w it would have been by (3 million/240 million populati $\times$ 30 =) 0.38 d. Between 1975–1982, about 80 mill kW in power plant orders were canceled, presuma causing LLE = 30 d.

APPLICATIONS OF THE CATALOG OF RISK

We offer a few suggestions for applying the cata of risks. One application is in personal decision maki For example, if a person is considering the purchase o small car, embarking on a jogging program, going on weight-reduction diet, choosing an occupation, or any numerous actions, it is useful to know the risks. Ri may not be the dominant factors in making a decisio but they are often important. However, the

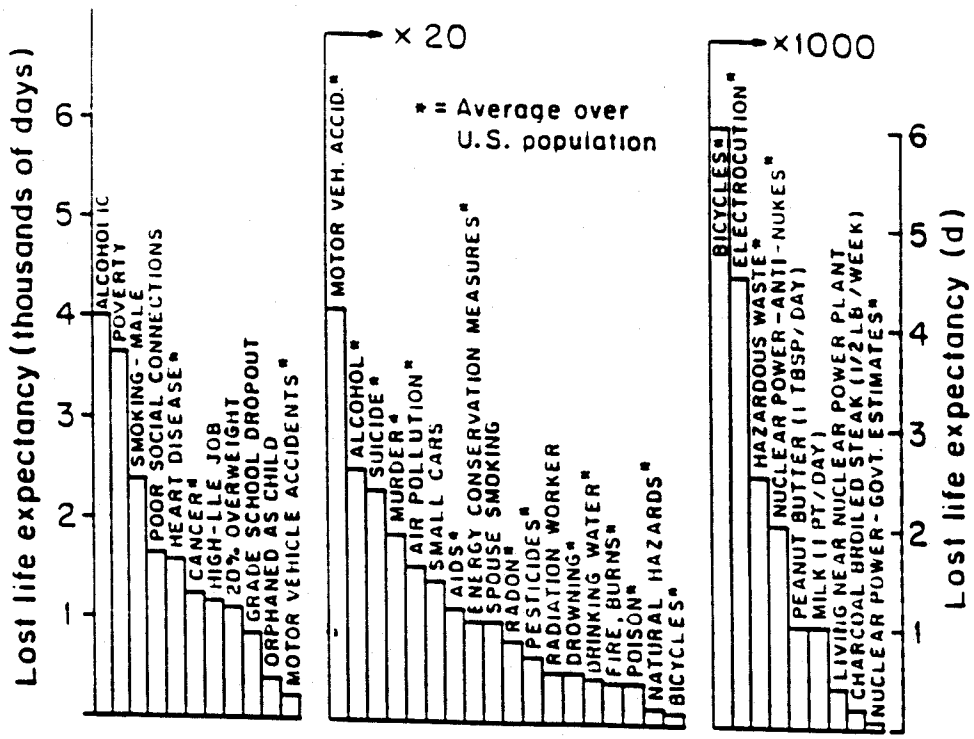

Fig. 2. Comparison of risks. Height of bar is LLE given in the text or from CR (1979) or OcR (1981). Asterisk designates average risk spread over the total U.S. population; others refer to risks of those exposed or participating. The ordinate scale is shown at the left. The heights of the bars are multiplied by 20 in the center section and by 1,000 in the right section. The first bar in each of these reproduces the last bar in the previous section, showing the effect of scale change.

we emphasize here is in helping to understand risks and to keep them in perspective. One aid to doing this is preparation of a bar graph (Fig. 2). Figure 2 includes data from CR and OcR as well as from this paper. Asterisks indicate risks averaged over the entire U.S. population while other items refer to those exposed or participating. Catastrophes refer to accidents involving a large number of deaths: the leading contributors (LLE in days in parentheses) are airline crashes (1), tornadoes (0.8), dam failures (0.5), major fires (0.5), hurricanes (0.3), earthquakes (0.15), and chemical releases (0.1).

Another approach to putting risks in perspective is to compare the amounts of exposure of major items that will give a risk equal to that of a minor item. For example, smoking one pack of cigarettes per day causes LLE = 2,250 d while nuclear power causes LLE = 0.05 d, a ratio of 45,000: one pack (20 cigarettes) per day is 45,000 cigarettes in 2,200 d. Thus, the risk of nuclear power is equal to the risk a regular smoker takes in smoking one extra cigarette every 2,200 d, or every 6 y. Similarly, the risk of nuclear power is equal to the risk of an overweight person increasing his weight by 0.6 g (0.02 oz.), or of using a small car rather than a mid-size car 1 d every 3 y.

Another approach is to compare risks of similar items. For example, a radiation worker at a nuclear plant gets more radiation exposure from Rn in his home than from his work.

This section on applications is not meant to be complete. It is only suggestive of some areas where these data may be useful.

REFERENCES

Auster, R.; Levenson, I.; Sarachek, D. The production of human health. J. Human Res. 4:411–436; 1969.

Berkman, L.; Syme, S. Social networks, host resistance, and mortality. Am. J. Epidemiol. 109:190–195; 1979.

Brenner, M. H.; Mooney, A. Unemployment and health in the context of economic change. Soc. Sci. Med. 17:1128–1135; 1983.

Chen, E.; Cobb, S. Family structure in relation to health and disease. J. Chronic Diseases 12:546–552; 1960.

Cohen, B. L. Perspective on occupational risks. Health Phys. 40:703–724; 1981. (Referred to in text as OcR.)

Cohen, B. L. Discounting in assessment of future radiation effects. Health Phys. 45:687–697; 1983.

Cohen, B. L. A survey of radon levels in U.S. homes: Results and correlating factors. J. Air Pol. Control Assn. 38:129–134; 1988.

Cohen, B. L.; Lee, I. S. A catalog of risks. Health Phys. 36:707–722; 1979. (Referred to in text as CR.)

Corneil, J. The great international disaster book. New York: Simon and Schuster; 1979.
Covey, L. S.; Wynder, E. Smoking habits and occupational status. J. Occup. Med. 23:541–549; 1981.
Crouch, E. A. C.; Wilson, R. Risk-benefit analysis. Cambridge, MA: Ballinger Publishing Co.; 1982.
Environmental Protection Agency. Unfinished business: A comparative assessment of environmental problems. Washington, D.C.: EPA; EPA Office of Policy, Planning and Evaluation Report; February 1987.
Evans, L. Accident involvement rate and car size. Accid. Anal. Prev. 16:387–405; 1984.
Evans, L. Driver fatality vs. car mass using a new exposure approach. Accid. Anal. Prev. 16:19–36; 1984.
Evans, L.; Wasielewski, P. Serious or fatal driver injury rate vs. car mass in head-on crashes between cars of similar mass. Accid. Anal. Prev. 19:119–131; 1987.
Fackelmann, K. A. More evidence ties smoke to artery disease. Sci. News 137:326; 26 May 1990.
Fahy, R. F.; Norton, A. L. How being poor affects fire risk. Fire J. 83(1):28–36; January 1989.
Ghana Health Assessment Project Team. A quantitative method of assessing the health impact of different diseases in less developed countries. Int. J. Epidemiol. 10:73–80; 1981.
Hadley, J. More medical care, better health. Washington, D.C.: Urban Inst. Press; 1982.
Hall, J. R. The latest statistics on U.S. home smoke detectors. Fire J. 83(1):39–41; January 1989.
Kissin, B.; Begleiter, H. Social aspects of alcoholism. New York: Plenum Press; 1976:99.
Kitagawa, E. M.; Hauser, P. M. Differential mortality in the United States: A study in socioeconomic epidemiology. Cambridge, MA: Harvard University Press; 1973.
Ledermann, S. Life expectancy at birth by occupational category in France. Population (Paris) 15:127–131; 1960 (in French).
Lew, E. A.; Garfinkel, L. Variations in mortality by weight among 750,000 men and women. J. Chron. Dis. 32:563–576; 1979.
Lew, E. A.; Garfinkel, L. Differences in longevity by sex, smoking habits, and health status. Trans. Soc. of Actuaries 39:107–125; 1987.
Lovejoy, C. O.; Meindl, R. S.; Pryzbeck, T. R.; Barton, T. S.; Heiple, K. E.; Kotting, D. Paleodemography of the Libben site, Ottawa County Ohio. Science 198:291–293; 1977.
McNeill, W. H. Plagues and peoples. Garden City, NY: Doubleday; 1976.
Melnick, S.; Cole, P.; Anderson, D.; Herbst, A. Rates and risks of diethylstilbestrol related clear cell adenocarcinoma of the vagina and cervix. N. Engl. J. Med. 316:514–516; 1987.
Myers, R. J.; Bayo, F. R. U.S. life tables for 1979–1981. Trans. Soc. of Actuaries 37:303–344; 1985.
Myers, D. K.; Werner, M. M. A review of the health effects of energy development. Nucl. J. Canada 1:14–24; 1987.
National Academy of Sciences. Committee on the Biological Effects of Ionizing Radiation. The effects on populations of exposure to low levels of ionizing radiation (BEIR III). Washington, D.C.: National Academy Press; 1980.
National Academy of Sciences. Committee on the Biological Effects of Ionizing Radiation. Health risks of radon and other internally deposited alpha-emitters (BEIR IV). Washington, D.C.: National Academy Press; 1988.
National Academy of Sciences. Committee on the Biological Effects of Ionizing Radiation. Health effects of exposure to low levels of ionizing radiation (BEIR V). Washington, D.C.: National Academy Press; 1990.
National Academy of Sciences. Bereavement. Washington, D.C.
National Center for Health Statistics. Mortality from selected causes by marital status. Rockville, MD: National Center for Health Statistics; Statistical Series 20: No. 8a; 1970.
National Center for Health Statistics. Selected vital and health statistics in poverty and nonpoverty areas of 19 large cities, United States, 1969–71. DHEW Publication No. (HRA): 76-1904; 1975.
National Center for Health Statistics. Geographic patterns in the risk of dying and associated factors, ages 35–74 years. Washington, D.C.: Department of Health and Human Services; Report No. (PHS): 80-1402; 1980.
National Council on Radiation Protection and Measurements. Influence of dose and its distribution in time on dose-response relationships for low LET radiations. Washington, D.C.: NCRP; NCRP Report No. 64; 1980.
National Oceanographic and Atmospheric Administration. Study of earthquake losses in the Los Angeles, CA area. Washington, D.C.: NOAA; 1973.
National Radiological Protection Board. Health effects models developed from the 1988 UNSCEAR report. Chilton, Didcot: NRPB; NRPB-R226; 1988.
National Safety Council. Accident facts. Chicago, IL: NSC; 1989.
Näyhä, S. Social group and mortality in Finland. Br. J. Soc. Prev. Med. 31:231–237; 1977.
Okrent, D.; Apostalakis, G.; Erdman, R.; Solomon, K.; Salem, S. Estimates of risks associated with dam failures. Los Angeles: University of California; Report UCLA-ENG-7423; 1974.
Ozkaynak, H.; Spengler, J. D. Analysis of health effects resulting from population exposures to acid precipitation. Environ. Health Pers. 63:45–55; 1985.
Partyka, S. Lives saved by seat belts from 1983 through 1987. Washington, D.C.: Department of Transportation; Report No. DOT HS 807324; 1988.
Ravenholt, R. T. Addiction mortality in the United States, 1980: Tobacco, alcohol, and other substances. Pop. Develop. Rev. 10:697–724; 1984.
Ravenholt, R. T. Leading preventable causes of premature death, U.S. mid-1980s. New York: American Council on Science and Health; 1987.
Registrar-General. Occupational mortality, 1970–1972. London: Her Majesty's Stationery Office; 1978.
Reif, A. E. Risks and gains. In: Vinger, P. F.; Hoerner, E. F., ed. Sports injuries: The unthwarted epidemic. Littleton, MA: PSG Publishing Co.; 1981.
Reinfurt, D. W.; Campbell, B. J.; Stewart, J. R.; Stutts, J. C. North Carolina's occupant restraint law: A three year evaluation. Chapel Hill, NC: UNC Highway Safety Research Center; Report No. HSRC-PR158; 1988.
Robin, E. Matters of life and death: Risks vs. benefits of medical care. San Francisco, CA: W. H. Freeman; 1984.
Royal College of General Practitioners. Oral contraceptives study. Mortality among oral contraceptive users. Lancet II-1977:727–731. Further analysis of mortality in oral contraceptive users. Lancet I-1981:541–546.
Rubenstein, H. S. Bee-sting diseases: Who is at risk? What is the treatment? Lancet I-1982:496–499.
Sagan, L. A. The health of nations. New York: Basic Books; 1987.
Silverstone, J. Obesity and social class. The Practitioner 202: 682–685; 1969.
Surgeon General. The health consequences of involuntary smoking. Washington, D.C.: Department of Health and Human Services; 1986.
Thompson, P. D.; Funk, E. J.; Carleton, R. A.; Sturner, W. Q.

Thompson. R. S.; Rivara. F. P.; Thompson. M. S. A case-control study of the effectiveness of bicycle safety helmets. N. Engl. J. Med. 320:1361–1363; 1990.

United Nations. The determinants and consequences of population trends. Population Studies 50:111–124; 1973.

United Nations. State of the world's children. 1984. New York: Oxford University Press; 1983:150.

United Nations Scientific Committee on the Effects of Atomic Radiation. Sources, effects, and risks of ionizing radiation. New York: United Nations; 1988.

Urquhart. J.; Heilmann. K. Risk watch. New York: Facts on File Publications; 1984.

U.S. Bureau of the Census. Low income areas in large cities. 1970 census of population. Subject Report PC(2)-9B; Washington. D.C.; 1973.

U.S. Bureau of the Census. Vital statistics of the United States. Sec. II. Part A; 1988.

U.S. Bureau of the Census. Supplement to the monthly vital statistics report: Advance reports. 1986. Vital and health statistics. Series 24; DHHS Document No. (PHS) 90-1953; 1990.

U.S. Department of Transportation. Fatal accident reporting system (FARS). Washington. D.C.: Report DOT HS 805953; 1981.

Weiss. R. The viral advantage. Sci. News 136:200; 23 September 1989.

Wigle. D. T.; Mao. Y. Mortality by income level in urban Canada. Ottawa: Ministry of Health and Welfare; 1980.

Wilkins. R. Health expectancy by local area in Montreal, a summary of findings. Can. J. Pub. Health 77:216–221; 1986.

World Bank. The effects of education on health. Washington. D.C.: World Bank; 1980.

World Resources Institute. World resources 1987. New York: Basic Books; 1987.

Thousands of Days of Lost Life Expectancy
1
2
3
4
5
6
ALCOHOLIC
POVERTY
SMOKING - MALE
POOR SOCIAL CONNECTIONS
HEART DISEASE*
CANCER*
HIGH-LLE JOB
20% OVERWEIGHT
GRADE SCHOOL DROPOUT
ORPHANED AS CHILD
MOTOR VEHICLE ACCIDENTS*
x 20
MOTOR VEH. ACCID.*
ALCOHOL*
SUICIDE*
MURDER*
AIR POLLUTION*
SMALL CARS
AIDS*
ENERGY CONSERVATION MEASURES*
SPOUSE SMOKING
RADON*
PESTICIDES*
RADIATION WORKER
DROWNING*
DRINKING WATER*
FIRE, BURNS*
POISON*
NATURAL HAZARDS*
BICYCLES*
* = Average over U.S. population
x1000
BICYCLES*
ELECTROCUTION*
HAZARDOUS WASTE*
NUCLEAR POWER - OPPONENTS*
PEANUT BUTTER (1 TBSP/DAY)
MILK (1 PT/DAY)
LIVING NEAR NUCLEAR POWER PLANT
CHARCOAL BROILED STEAK (1/2 LB/WEEK)
NUCLEAR POWER - GOVT. ESTIMATES*
1
2
3
4
5
6
Days of Lost Life Expectancy

Fig. 2