Cost-Benefit Analysis and the Environment
Further Developments and Policy Use

PART III

Chapter 15

Health valuation

The valuation of health risks is a long standing area of both research and policy application. Even so, increasing evidence of the global burden of disease and especially the role of environmental pollution as a determinant of this burden has added a further urgency to this work. Considerable strides have been made in recent years in terms of clarifying both the meaning and size of the value of statistical life (VSL). One of the main issues has been how to "transfer" VSLs taken from e.g. from country-to-country or where life expectancy of those people who are the object of policy and investment project proposals differs. Needless to say, this still requires that applications are done with care and judgement. In some areas, the literature offers firmer guidance here than in others. Notably, age may or may not be relevant in valuing immediate risks – the literature is arguably ambiguous with regards to the empirical relationship. That said, in terms of practical guidelines, the empirical record has been important in translating findings in base or reference levels for health values for use of policy or project appraisal.

15.1. Introduction: the importance of health effects in CBA[1]

Environmental policy affects human health in a number of ways. First, by reducing environmental risks to lives, it may "save lives", i.e. reduce premature *mortality*. Second, it may improve the health of those living with a disease, e.g. a respiratory illness. This is a *morbidity* benefit, relating to physical health. Third, it may reduce the stresses and strains of living and thus improve *mental health*. By and large, environmental economics has focused on the first two types of benefit by evaluating how environmental policies improve provision of these benefits (such as actions to improve air quality in urban areas) or how projects diminish these benefits (such as energy or road transport investments). Less attention is paid to the third effect. That said, some would argue that these effects could be captured by individuals' willingness-to-pay to reduce stress – e.g. from excessive noise. The recent focus on subjective well-being (and its links to environmental quality as a determinant) has made more of this link to mental health (see Chapter 7).

A striking example of what is at stake is illustrated by the Global Burden of Disease project (see, for example, Murray et al., 2016). According to its most recent assessment, about 6.5 million deaths worldwide in 2015 were attributed to exposure to all air pollution.[2] In total, environmental risks (which also includes unsafe water and sanitation as well as household and occupational exposure to hazardous substances) is said to account for 29% of total mortality in that year. This Chapter begins by explaining how such physical magnitudes can be estimated. These are the bedrock of any subsequent economic assessment. The procedure here is to take an "objective" measure of risk arising from some change in an environmental variable, here a measure of air quality such as the concentration of particulate matter in the ambient environment. This is shown as a *dose-response function* or *exposure response function* and is used to estimate numbers of premature mortalities.

From an economic perspective, what really matters is the value that people place on these burdens. Section 15.3 sets out the relevant valuation concepts and identifies how practitioners have sought to empirically estimate these parameters. By and large, the procedure for valuing risks to life, i.e. *a mortality risk*, have involved an estimation of the willingness-to-pay (WTP) to secure a risk reduction arising from a policy or project, or the willingness-to-accept (WTA) compensation for tolerating higher than "normal" risks. The procedure involves taking the risk change in question and dividing this into the WTP (or WTA) for the risk reduction, to secure a "value of statistical life" (VSL).[3] However the VSL estimated, mortalities estimated using dose-response functions can be multiplied by that VSL to give an aggregate measure of the burden.

Of course, a proposal for a policy or a project entails *valuing changes* in such burdens. There is increasing evidence, however, that when a health benefit is present as an impact of a policy or investment project, it often appears to "dominate" cost-benefit studies. If so then it matters a great deal that the underlying theory and empirical procedures are correct. Much of the remainder of this Chapter then is concerned with a number of the debates surrounding the *validity* of VSL. Notable here is how estimates of WTP for risk reduction pass key tests such as "scope" which means that higher risk changes should be associated with higher WTP. Whether studies pass or fail such tests can be used to screen the quality of the empirical record, with OECD (2012) being one prominent example of this. This screening is often undertaken in order to establish a standard value for the VSL, which can be applied to mortality risks across countries; although perhaps adjusting for income differences. Others would take this further arguing that the VSL is heterogeneous, not just in income but in other policy relevant socio-demographic characteristics, notably such as age. The concern here is that a standard VSL "for all" might "overstate" health burdens given the (higher) average age of those primarily affected by environmental problems such as air pollution. Notwithstanding important questions about equity, whether this concern has a firm basis in empirical evidence has been the subject of some debate. And while the focus is primarily on mortality risk in this Chapter, valuing non-fatal health burdens – i.e. morbidity – is also relevant and discussed in the penultimate section.

15.2. The global burden of air pollution

The physical sources of health risk in urban and rural environments are both numerous and diverse. These include water-borne sources of illnesses and disease as well as health effects arising from changing temperature as a result of climate change. There is increasing interest also in the way in which the lack of access to green space might enhance human health (in both physical and mental health dimensions). It is fair to say, however, that particular attention has centred on the link between air pollution and health, notably particulate matter and especially tiny particles such as $PM_{2.5}$ (particulate matter of 2.5 microns or less in diameter).[4] These substances in ambient air across major cities, urban areas as well as rural locations are now seen as being implicated in large numbers of deaths around the world. The exact causes of mortality to which $PM_{2.5}$ contributes are varied but notably include heart disease and stroke as well as lung cancer, respiratory infection and chronic respiratory disorders.

This has far-reaching implications for environmental policy as well as investments in sectors such as transport and energy. For example, Hamilton et al. (2014) ties this to questions about the benefits of climate policy by looking at the possible size of the *health co-benefits* from mitigating greenhouse gases in energy projects. They estimate the value of air pollution mortality per tonne of CO_2 to be more than USD 100 for high income countries and USD 50 for middle income countries. Such numbers can be compared with the carbon prices in Chapter 14, for example.

The starting point for much of this economic analysis is the work of the Global Burden of Disease project (see Murray et al., 2016, Vos et al., 2016 and also GBD, 2013). This assesses ambient $PM_{2.5}$ by monitoring concentrations on the ground along with other approaches such as satellite observations. This complementary approach helps build a fuller picture of human exposure across e.g. urban areas in a more comprehensive way than has been possible in the past. The burdens are shifting too, as some regions experience decreasing exposure to $PM_{2.5}$ between 1990 and 2013 (much of Europe and the United States as well as parts of South East Asia) while other regions experience increasing exposure (sometimes substantially) such as South and Far East Asia, parts of Southern Africa and South America.

While concentrations of $PM_{2.5}$ measured by micrograms per cubic metre of air (μg per m^3) vary across locations (due to natural and human factors), the physical impact on premature or excess deaths is calculated in a number of steps for which, typically, the first is the calculation of the relative risk, *RR*, of death. This number compares the change in the risk of death from a given unit change in $PM_{2.5}$. For example, if the *RR* = 1.048 per 10 μg per m^3, this means that the risk of dying as a result of exposure to a 10 μg per m^3 change concentration of $PM_{2.5}$ is 1.048 times higher than previously.

RR therefore specifies the relationship between pollution concentrations (the dose) and the response (health effect). The results of existing statistical studies of the association between exposure to air pollution and human health effects (such as mortality risks) across populated areas allow estimation of *RR* as:

$$RR = e^{\beta(C_1 - C_0)}$$

where C_1 is the current level of pollution and C_0 is some reference level. The parameter β is a risk factor reflecting the severity of the health risk.

Critically this relative risk is used to calculate the "attributable factor" or AF: the proportion of fatalities (in a given year) which are attributable to exposure to pollutant levels above the reference level. This *AF* is calculated as follows:

$$AF = (RR - 1) / RR$$

So for example, for C_1 = 20 μg per m^3, C_0 = 10 μg per m^3 and β = 0.0047 then *RR* = 1.048. In this case, *AF* = 0.046. This means that 4.6% of total deaths are attributable to exposure to $PM_{2.5}$ at levels of 20 units rather than the reference level of 10.

For illustrative purposes, assume the geographical area of interest is a city with a population of 5 million people, all facing mortality risks due to air pollution. The death rate (all causes) is 9 per 1 000 people. So the total number of deaths in the city in any one year is expected to be *B* × *POP* = 0.009 × 1m = 45 000 deaths.

$$\Delta H = AF \times B \times POP$$

The *AF* estimated indicates how many of these total deaths are attributable to air pollution (strictly speaking, how many deaths because pollution concentrations 10 units above the reference level, in this example). Multiplying *AF* by 45 000 indicates that 2 070 deaths are caused by air pollution in this example.

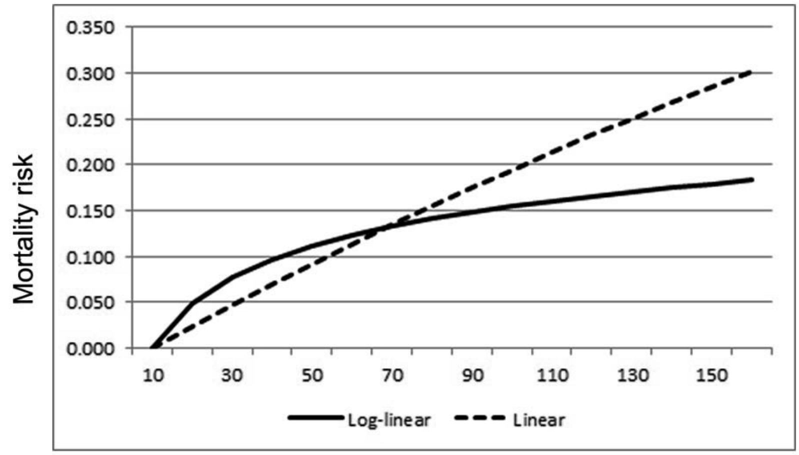
These calculations assume that health impacts increase linearly with pollution concentrations (i.e. the broken line in the Figure 15.1. There is evidence, however, that the number of *extra* cases tails off as pollution concentrations increase, at least for certain types of cause of death, such as heart disease (i.e. the unbroken line in the same figure). The previous calculations do not capture this but the following expression does:

$$RR = (C_1 / C_0)^{\gamma}$$

where Figure 15.1 illustrates this for the case where γ = 0.0073. The GBD study, as well as subsequent work by World Bank (2016) and Hamilton et al. (2014), also employ this latest evidence on this relationship. This allows a more realistic assessment of deaths which are likely to occur at higher levels of $PM_{2.5}$ concentration, such as those which prevail in many mega-cities in the developing world.

Table 15.1 reports some of the results of World Bank (2016) for 8 countries. It indicates ambient $PM_{2.5}$ concentrations and subsequent total deaths, estimated using a dose-response function along the lines just discussed. For each of these countries, the first row of reported numbers is the value for 2013. The second (italicised) row is the change between 1990 and 2013. The table indicates that ambient $PM_{2.5}$ declined over the period in 5 of these 8 countries. However, in The People's Republic of China and India in particular,

Figure 15.1. **The relationship between PM exposure and mortality risk**



Source: Authors' calculation from assumed values and functional forms.

Table 15.1. **The health burden of ambient $PM_{2.5}$ by selected countries**

1990-2013

		Ambient $PM_{2.5}$ concentrations (g/m^3)	Total $PM_{2.5}$ deaths	Economic well-being losses (% of GDP)
Brazil	2013	16.5	62 246	3.0%
	Change 1990-2013	*+6.8*	*+2 640*	*+0.00*
The People's Republic of China	2013	54.4	1 625 164	10.0%
	Change 1990-2013	*+15.1*	*+106 222*	*+3.00*
India	2013	46.7	1 403 136	8.0%
	Change 1990-2013	*+16.4*	*+359 954*	*+1.00*
Russian Federation	2013	14.2	104 379	8.0%
	Change 1990-2013	*-5.4*	*-9 365*	*-1.00*
France	2013	14.0	21 138	3%
	Change 1990-2013	*-8.7*	*-6 326*	*-2.00*
Germany	2013	15.4	41 485	5%
	Change 1990-2013	*-14.4*	*-29 651*	*-5.00*
UK	2013	10.8	19 803	3.2%
	Change 1990-2013	*-8.9*	*-25 650*	*-5.70*
US	2013	10.8	91 045	2.8%
	Change 1990-2013	*-5.7*	*-36 195*	*-2.50*

Notes: Italicised values are changes. For ambient PM2.5 (column 3), this indicates how many points higher (or lower) concentrations were in 2013 compared with 1990. Column 4 indicates how the number of deaths has changed. Columns 5 and 6 relate economic damages to GDP. For example, in 2013, economic well-being losses are 10% of GDP. In 1990, this burden was 7%. The change is therefore described as +3.00 (3 percentage points higher).
Source: Adapted from World Bank (2016).

$PM_{2.5}$ concentrations increased, where in the latter this results in more than a 25% increase in annual mortality (in 2013 compared with 1990).

The contribution of studies such as World Bank (2016) is to work out what this means for $PM_{2.5}$ as an economic burden. This requires a link between health effects and human well-being, by estimating the monetary value of these health damages (*D*):

$$D = P \times \Delta H$$

For mortality risks, *P* typically is some measure of the value of statistical life (VSL), discussed in more detail in section 15.3. In World Bank (2016), a reference value of USD 3.8 million

(2011, prices) is assumed. This, in turn, is based on a study by OECD (2012), which sought to estimate (using the available empirical record) a reference value for VSL for high income countries. In the World Bank study, this value is then adjusted according to income differences across the countries. This is explained in more detail in Section 15.4 below.

Table 15.1 illustrates these monetary values in its final two columns. It is notable that even for those countries where $PM_{2.5}$ concentration declined over the study period, the economic burden in terms of well-being losses remained significant in 2013 as a percentage of country GDP. For Germany, this is 5% of GDP in 2013 (down from 10% of GDP in 1990). In Brazil, India and The People's Republic of China, ambient $PM_{2.5}$ increased over the period. This translates into a substantially higher mortality burden. For example, $PM_{2.5}$ mortality was over a third higher in 2013 in India than in 1990. The loss of economic well-being – defined in terms of how much those at risk would be willing to pay to eliminate these health risks – are consequently increasing too, reaching 8% and 10% of GDP in India and The People's Republic of China respectively.

Table 15.2 summarises the findings of this study for regions of the world for all air pollution, which includes ambient $PM_{2.5}$ exposure as well as ambient ozone exposure and indoor air pollution. It also describes this burden for ambient $PM_{2.5}$ only. In each case these values are reported as a percentage of regional GDP. As can be seen, the burdens are high, although not always almost all associated with ambient $PM_{2.5}$ as the cases of East Asia and Pacific, Latin America and Caribbean, South Asia and Sub-Saharan Africa indicate.

Table 15.2. **The economic burden of air pollution by region**

As a Percentage of GDP, 2013

	Based on VSL		Based on labour income	
	All air pollution	$PM_{2.5}$	All air pollution	$PM_{2.5}$
East Asia and Pacific	7.5	4.5	0.25	0.15
Europe and Central Asia	5.1	4.8	0.13	0.12
Latin America and Caribbean	2.4	1.5	0.13	0.09
Middle East and North Africa	2.2	2.0	0.14	0.13
North America	2.8	2.4	0.11	0.10
South Asia	7.4	3.1	0.83	0.39
Sub-Saharan Africa	3.8	1.4	0.61	0.23

Source: Adapted from World Bank (2016)

Basing this assessment on the VSL indicates the magnitude of the *loss of well-being* as a result of these health burdens. Relating these values to GDP is for comparative purposes: that is, to get a sense of the scale of these well-being losses rather than to claim that GDP straightforwardly could be higher were it not for the burden of air pollution. That said, it is highly likely that such burdens do impact negatively on the economy. To illustrate this, World Bank (2016) estimates the impact on income and productivity and so a more narrow emphasis of the burden on the economy. This is estimated as the loss in the (present value of) foregone earnings as a result of premature mortality. Not surprisingly, Table 15.2 notes that these magnitudes are far smaller than the corresponding well-being losses. Nevertheless, for South Asia and Sub-Saharan Africa in particular, this value is not trivial; nor is it attributable to ambient $PM_{2.5}$ only as indoor air pollution appears to contribute significantly to these economic losses too.

This "static" assessment may not reflect full impact of these health burdens on the economy. For example, this lost productivity translates into lost output, some of which in turn might have invested productivity and the returns on those actions consumed at a later date. Put another way, there is a dynamic impact of these mortality risks which may also need to be considered. For example, in the cases of both The People's Republic of China and Europe, (respective) contributions by Matus et al. (2012) and Nam et al. (2010) show that this matters empirically. As an illustration, the former study shows that the (dynamic) costs of air pollution were around 6% to 9% of GDP for The People's Republic of China over the period 1995 to 2005. This is at least one and half times greater than previous studies which looked only at these costs, over a comparable time period, from a "static" perspective.[5]

15.3. Valuing life risks: the VSL concept

The previous section ended with a discussion of the value of mortality risk, in the context of the global burden of air pollution. It is important to take a step back and consider the conceptual basis for this monetary valuation as well as the empirical issues which arise from the practical estimation of these values.

Starting with the conceptual details, the Annex to this chapter shows the standard derivation of the VSL expression for the simplest case. The final equation is:

$$VSL = \frac{dW}{dp} = \frac{u_a(W) - u_d(W)}{(1-p).u_a'(W) + p.u_d'(W)}$$

where W is wealth, p is the probability of dying in the current period (the "baseline risk"), (1-p) is the probability of surviving the current period, u is utility, "a" is survival, and "d" is death. The utility function u_d allows for bequests to others on death. The numerator thus shows the difference in utility between surviving and dying in the current period. The denominator shows the marginal utility of wealth (which is usually measured empirically as income) conditional on survival or death. The expected relationships between VSL, p, W and expected health status on survival are discussed in Annex 15.A1.

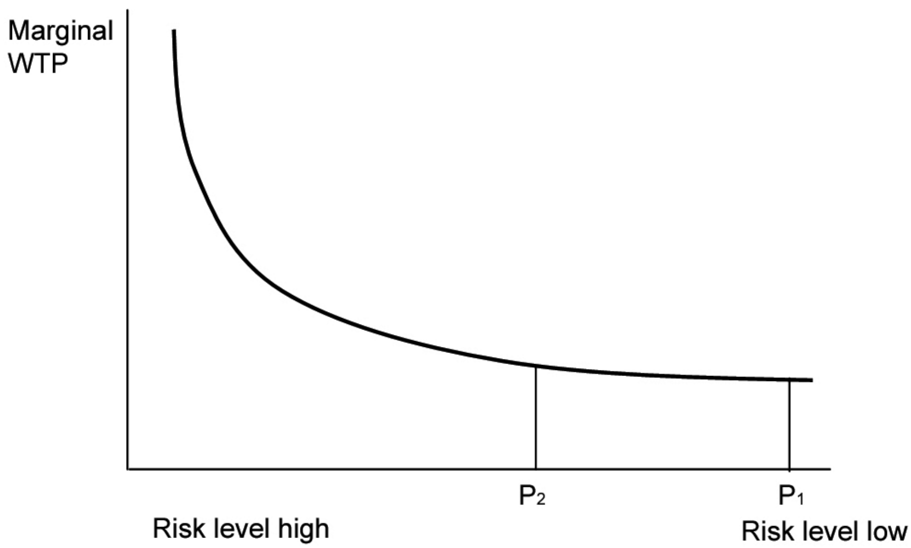
Figure 15.2 illustrates the link between WTP and risk levels. VSL is a *marginal* WTP and hence Figure 15.2 shows marginal WTP against the risk level. The status quo risk level is usually referred to as the initial or baseline risk level. Policy usually involves *reducing* risks so as the risk level declines so does marginal WTP, as shown in Figure 15.2, and as risk rises so marginal WTP is expected to rise.

Suppose the policy measure in question reduces risk levels from P_2 to P_1 in Figure 15.2. Then the WTP for that risk reduction is seen to be equal to the area under the marginal WTP curve between P_2 and P_1. Notice that marginal WTP may be fairly constant at low levels of risk (to the right of the diagram). Small differences in the initial (baseline) risk level are therefore usually assumed to have little effect in VSL studies.[6]

It tends to be assumed that the quality of the period survived affects WTP, i.e. WTP to reduce risks should be higher if the individual anticipates being in good health (apart from the risks in question), and lower if the individual expects to be in poor health. The equation implies that WTP rises with wealth since (a) it is assumed that the marginal utility of wealth is greater for survival than as a bequest when dead, and (b) there is aversion to financial risk. The former makes the numerator increasing in wealth.

As an example, suppose a policy promises to reduce risks from 5 in 10 000 to 3 in 10 000, a change of 2 in 10 000 (ΔRISK). Suppose that the mean WTP to secure this risk reduction is USD 750. Then the VSL is usually computed as: $\frac{WTP}{\Delta RISK} = \frac{750 \times 10\,000}{2}$, and so the VSL would be around USD 3.8 million.

Figure 15.2. **Risk and willingness-to-pay**



There are a variety of ways in which, as a practical matter, VSL might be estimated. Broadly speaking, these might be distinguished as to whether the underlying valuation concept is WTP to secure a risk reduction arising from a policy or project, or the WTA compensation for tolerating higher than "normal" risks. Studies of the former have involved use of stated preference techniques (see Chapters 4 and 5). But it also might involve looking at revealed behaviour such as avertive expenditures (see Chapter 3). The latter have involved hedonic wage risk studies (see Chapter 3).

One finding is that hedonic risk studies appear routinely to produce higher values than stated preference studies in context of public transport accidents. There are at least two reasons that might explain this. First, occupational risks tend to be higher than public risks. If valuations are reasonably proportional to risk levels, as the theory predicts, then one would expect higher values from occupational studies. Second, hedonic risk studies measure WTA, not WTP. While the relationship between WTP and WTA is still debated (see Chapter 4), a number of reasons have been advanced for supposing that WTA will exceed WTP, perhaps by significant ratios. One suggestion is that, whilst interesting, the hedonic wage studies do not "transfer" readily to the context of public transport accidents.

As an illustration of studies which have been applied to the transport accident context, Table 15.3 describes some VSL studies for Sweden drawing on a review by Hultkrantz and Svensson (2012). This reveals a range of estimates from EUR 0.9 million to EUR 6.2 million (2010 prices). In principle, estimates could be used to guide recommendations for an official VSL to be used in CBA. In this context, Hultkrantz and Svensson report that the official value for road traffic accidents in Sweden is just over EUR 2 million, which in the context of the table would put it towards the lower end of the range. Some of these studies, however, are estimating different things using different methods. For example, some estimate VSL for risks which are private goods while others estimate risks involving public goods. Some of these studies use revealed preference methods while others use stated preference. Asking what might be a defensible "consensus" value for a VSL, involves, in turn, asking further probing analytical questions of the empirical record.

Table 15.3. **Selected VSL Studies in Sweden from 2005 to 2010**

Publication	Study Year	Method	Type of good (and risk)	VSL, million euro (2010, prices)
Andersson	2005	RP	Road (private risk)	0.9
			Road (private risk)	1.6
Hultkrantz et al.	2006	SP	Road (private risk)	6.2
			Road (public risk)	2.4
Svensson	2009	RP	Seat belt (private risk)	2.3
			Cycle helmet (private risk)	4.0
		SP	Road (public risk)	2.3
			Road (public risk)	3.4

Source: Adapted from Hultkrantz and Svensson (2012).

This is essentially what OECD (2012) does in undertaking a meta-analysis of a large number of studies of mortality risk valuation drawn from around the world. Common to each is the use of stated preference (SP) surveys to estimate the VSL (specifically by eliciting respondent WTP for reductions in mortality risk). Broadly speaking, the intentions of this study are two-fold (but related). One is an analytical objective to understand better the sources of variation in VSL across original studies across countries. The other is to compute base values of VSL, which can be used within countries as well as used to transfer values across countries. Such base values can be adjusted in a number of ways to suit better the circumstances of the VSL context and country in which estimates are applied. But the point is that the prior meta-analytical probing of the empirical record provides a consistent basis for such adjustments.

Turning to results of OECD (2012), Table 15.4 illustrates that the mean VSL for the full sample of 856 VSL estimates was USD 7.4 million (2005 prices).[7] The Table also indicates that the median value was much lower, at about USD 2.4 million. As this publication explains, it is important to look beyond such headlines. One reason for this is that the range of mean VSL across these studies is large. To control for undue influence of the outer reaches of this range, the trimmed sample in Table 15.4 removes the highest and lowest outliers. Another reason is that some of the studies on which even this trimmed sample is based are likely to be of a higher quality than others. Here, "high quality" refers to satisfying criteria on what is broadly considered to be good practice. The OECD meta-study uses four such criteria for screening studies. First, is adequate information provided on the value of a risk change? Second, is there an adequate sample size (specifically, are main samples greater than 200 and sub-samples no smaller than 100)? Third, is the sample representative of a broader population? Fourth, did the authors of these various think their estimate was suitable for inclusion? These are reasonably generous criteria but when applied reduce the number of studies in this screened sample to 405.

Table 15.4 reports the mean VSL based on this screened sample to be about USD 3.1 million (in 2005 prices). For the subset of studies from OECD countries, the mean value is just under USD 4 million and about USD 4.9 million for EU27 country studies. As previously mentioned, these findings have a practical intent. OECD (2012) suggests a base-value of USD 3 million for use in OECD countries (2005 prices). This is based on the median VSL indicated in Table 14.4, thereby avoiding the extremes of the range within a sample. The report also suggests a lower and upper bound of -50% and +50%, reflecting likely sizes of transfer errors. So for these OECD countries, the range is USD 1.5 to USD 4.5 million (in 2005 USD). A slightly higher base value is recommended if the focus is the EU27 only: i.e. USD 3.6 million, again the median VSL for that sample in Table 15.4.

Table 15.4. **Mean VSL across studies**

USD million, 2005 prices, standard error in parentheses

	Full sample	Trimmed sample	Quality screened sample		
			All countries	OECD countries	EU27 sample
Mean VSL	7.42 *(0.88)*	6.31 *(0.30)*	3.12 *(0.25)*	3.98 *(0.29)*	4.89 *(0.44)*
Median VSL	2.38	2.38	1.68	3.01	3.61
Sample	856	814	405	261	163

Note: These values are weighted averages where each mean VSL estimate in each individual study is weighted by the inverse of the number of observations from each SP survey; Trimmed sample refers to removing the highest and lowest 2.5%.

Source: Adapted from OECD (2012).

15.4. VSL issues and debates

The previous section set the scene for VSL by briefly explaining the concept and moving swiftly through to discussing how base or reference values of VSL have been constructed from the empirical record. A critical question is: what explains these values? Clearly the answer to this question is important to practical matters such as what the magnitude of the VSL to use in CBA in a particular country in a particular risk context should be. But another reason why this question is so important is that it throws light on a number of conceptual and empirical puzzles that have also characterised debates about VSL. A number of these are discussed in turn, in what follows.

15.4.1. The sensitivity of VSL to risk levels

The theory of the VSL requires that WTP varies directly with the size of the policy-related risk: i.e. the risk change brought about by the policy or project in question, and for which the VSL is usually sought.[8] Indeed, it is widely considered that sensitivity to absolute risk is a basic test of the validity of any preference-based technique for measuring the VSL. Empirical investigation of whether what is expected in theory is actually borne out in practice has focused on a number of elements of this relationship.

An early review was Hammitt and Graham (1999) which looked at contingent valuation studies of WTP for risk reduction. In particular they tested for two predicted relationships: (a) that WTP should vary directly with the size of the risk reduction, and (b) for low probabilities (probability being their chosen measure of risk), WTP should be virtually proportional to the change in risk. Thus, if WTP for a change in risk ΔX (where X is small) is W, WTP for $\alpha\Delta X$, should be αW. They also look at "baseline risk", i.e. the level of risk from which ΔX deviates (the background risk as outlined above).

Of the 25 studies they reviewed (up to 1998), only 10 contain sufficient information to test scope sensitivity within sample (internal validity). And of those 10 studies, most confirm the first hypothesis that WTP varies with risk reduction, but not the second. Proportionality is not observed. Even in the former case, a significant minority of respondents report the same WTP regardless of the size of risk change, or ΔX. External validity (across sample scope tests) assessments showed a similar pattern, but with even the first hypothesis receiving only weak support.

OECD (2012) brings these findings further up-to-date, although notably the conclusions stay broadly the same. The size of the risk change is negatively correlated with VSL in the full sample, thus violating the expectation in theory that VSL should be invariant to whether respondents were offered a small or large risk reduction. Even screening, however, for those studies where there was clear scope *in*sensitivity and excluding these from the analysis, this finding holds. Again what this indicates is that there is no proportionality and, as a result, VSL varies depending on the size of risk change "offered" to respondents.

That this problem appears to have persisted is an important finding, and naturally shifts attention to questions about why scope insensitivity may occur. For stated preference studies, the notable problems in communicating low risk levels to respondents, given that such low risks typically define environmental contexts.[9] Visual aids, and better communication more generally of these risk changes in surveys, might help although the persistence of the finding indicates that this is unlikely to resolve the issue. On the one hand, there is evidence that bad quality studies amplify the problem. On the other hand, even in high quality studies there is evidence that the problem persists to some degree, and so another issue might be "bad quality" human subjects (e.g. respondents in SP studies). That is, it may be that the nature of risk changes associated with environmental policy are simply too small for people to identify with in ways that might otherwise reasonably be expected. This echoes the behavioural debates playing out in SP research more generally (see Chapter 4).

The implications of the risk scope sensitivity analyses for environmental CBA are not easy to determine on the balance of such arguments, although perhaps suggest that caution and awareness of the issue in conducting sensitivity is needed at a minimum.

15.4.2. VSL and the income elasticity of willingness-to-pay

WTP should vary directly with income. Indeed, it is widely considered that sensitivity to income is the other basic test of the validity of any preference-based technique for measuring the VSL. Most studies find that this WTP does indeed vary with income. Apart from the requirement that WTP should vary with income as a theoretical validity test, the link between income and WTP is of interest for other more practical reasons. Often in valuation exercises there is the need to account for rising relative valuations of benefits and costs over time. This means ascertaining the likelihood that a given benefit or cost is likely to have a higher (or lower) real unit WTP in the future. For example, suppose that the willingness-to-pay to save a statistical life rises faster than the rate of inflation (which is always netted out in CBA). Then it would be correct to include that rising real valuation in the CBA formula over time.

Another reason is that studies such as OECD (2014) and World Bank (2016) make use of these findings for transfer exercises across space (e.g. country to country): that is, to "gap-fill" VSL estimates, from countries which have these data to those which do not and involves following the widely cited formula (see also Chapter 6):

$$VSL_i = VSL_{OECD} \times \left(\frac{Y_i}{Y_{OECD}} \right)$$

In this formula, *VSL* is an average of (quality scrutinised) studies from high income members of OECD. This VSL_{OECD} is simply adjusted downwards for use in countries where people have lower incomes (than the per capita average of these OECD countries) and, vice versa, for those with higher per capita incomes. Specifically, the World Bank study uses a more general form:[10]

$$VSL_i = VSL_{OECD} \times \left(\frac{Y_i}{Y_{OECD}} \right)^b,$$

where "*b*" is the income elasticity of *VSL*. This indicates how *VSL* (based on *WTP* to reduce health risks) varies with per capita income levels.

What value then should "*b*" take for this rudimentary value transfer?

The World Bank study uses a value for high-income to high-income country transfers *b*=0.8 (see also OECD, 2014). What this means is that if per capita income increases by 5% then *VSL* increases by 4% (i.e. 5% × 0.8). A finding in OECD (2012) supports this assumption as it estimates b to lies in the range 0.7 to 0.9, at least when considering the screened subset of what it considered to be higher quality studies.

For high-income to low-income transfers, World Bank (2016) assumed that *b*=1.2. What this means is that if per capita income increases by 5% then *VSL* increases by 6% (i.e. 5% × 1.2). This is a little bit different to the value of this parameter recommended in Hammitt and Robinson (2011). That review concludes that for transferring *VSL* estimates from high to low income countries a value of "*b*" of 1 is the most defensible assumption (see for example, Roy and Braathen, 2017).

Using sensitivity analysis in such circumstances is probably a sensible approach and World Bank (2016), for example, uses a range of 1.0 to 1.4 for assumed values of *b* for high- to low-income transfers, and a range of 0.6 to 1.0 for high- to high-income transfers.

15.4.3. The context of VSL

Assuming that VSL estimates are accepted for policy purposes, interesting issue arise about what its size is and whether a VSL estimated in one context, say road accidents, be applied to another context, say environmental pollution? Various countries adopt single values for the VSL and use them in policy appraisal. Usually, estimates are not varied by context but clearly there is a question about the extent to which the transferability of single values is valid.

Chilton et al. (2002, 2004) tested for the effect of risk context on valuations. These studies directly sought valuations of risks in rail and fire contexts and air pollution contexts relative to risks in road accidents. The general conclusion is that context makes little difference. Perhaps, at best, domestic fires are valued about 10% less than a road accident, probably reflecting the degree of control individuals feel they have over domestic fire. For air pollution values, the finding is that these are valued at 10% more than a road accident, so again, context appears not to have a significant effect on valuation. OECD (2012) appears to confirm those earlier findings for its larger pool of studies. There appears little evidence looking across the empirical record that context matters substantially.

One other specific aspect of these concerns about context is whether people value so-called "dread risks" differently. The idea here, for example, is the belief that there may be a higher WTP to avoid cancers than other diseases. This is because of the "dread" effect of such a serious illness. If so, there is a "cancer premium" to consider in estimating a risk context appropriate VSL. This is especially relevant in the case of air pollution, especially $PM_{2.5}$ which has been implicated in this particular health pathway.

Hammitt and Liu (2004) find evidence that there is a cancer premium which they estimate to be about one-third, i.e. VSL for avoiding a cancer risk is 1.3 times that of a VSL for some other disease. OECD (2012), however, does not find substantial support for this across the broader literature (although the sample size for this test remains small). However, inferences about this might be sought from the literature on WTP to avoid non-fatal cancers (NFCs). The indications here are that these values fractions of a VSL, but with values being proportional to some "dread" factor. If so, then it is not implausible to think that VSL might vary with type of disease causing death.

15.5. Heterogeneity and VSL: An age-related VSL?

Much debate in the VSL literature has focussed on its apparent heterogeneity. That is, the VSL appears empirically to differ across people depending on socioeconomic and demographic characteristics. By contrast, policy applications typically rely on "standard values": that is, estimates of the VSL which is invariant across different groups whose mortality risks are being affected by policy. One prominent venue in which this debate plays out is the way in which the age of individuals may (or may not) matter in relation to the VSL of people over their lifetime. While the typical practice is to apply standard values for the VSL regardless of age (or other individual characteristics), once the VSL discussion is placed in the context of environmental policy, such as air quality management, it is quite possible that age matters in a potentially significant way. This is because pollution control policy tends to "save" lives of older people, or, to put it another way, pollution has the effect of "harvesting" the older population.

Two risk contexts need to be distinguished in the CBA of health impacts arising from environmental policy: immediate and future risks. For *immediate risk*, the WTP to avoid that risk which could occur "tomorrow" or, at least, in the next few months or years, i.e. *acute risks*. But there are also *latent risks*, i.e. situations in which exposure now does not produce death until a much later period. Of course, the reality is that policy context is likely to be one of both immediate and future risks. In the air pollution example, the risk may well be immediate for older people since it is known that it is older people who tend to be most affected by air pollution, i.e. the risks they face are still acute. But for younger people, while immediate benefits are considerably less, the benefits of reducing pollution will accrue to this younger group when they are much older. This is not always the case of course and it is important to note as well a separate issue. Mortality risks may have a significant incidence among children – the issue of valuing children's (statistical) lives. Bringing the effects on children into the domain of CBA is potentially important, with a default position being to use the adult valuations of "own" life risks for the risks faced by children.

The question naturally arises as to whether someone aged, say, 70 years of age has the same WTP to avoid a mortality risk as someone of e.g. 35 years of age. More critically, environmental policies may save a disproportionate number of lives in the "very old" category, i.e. reducing mortality risks which imply extending (statistical) life months, weeks or even days later without the policy. The issue, then, is what weight should be attached to such risk reductions in a CBA.

Age is usually thought to have two potentially offsetting effects in relation to the VSL: (a) the older one gets, the fewer years are left so the benefit of any current reduction in mortality risk declines – that is, it would be expected for VSL to decline with age; (b) the opportunity cost of spending money on risk reduction declines as time goes by because savings accumulate, so WTP for risk reduction may actually rise with age. As Aldy and Viscusi put it: as an individual ages, life expectancy decreases (by definition) but economic resources that the individual has may vary as well. Given these possibly offsetting effects, the question is what reasonably might be expected with reference to either theoretical precepts, empirical evidence or some mixture of the both.

Theoretically, the literature suggests that WTP should vary non-linearly with age, with an upside-down "U" curve that probably peaks at some point around middle-age (Shepard and Zeckhauser, 1982; Arthur, 1981). A lot of debate understandably has surrounded whether this relationship is robust. Some of this debate has sought to probe further the theoretical grounds for this form of non-linearity (Johansson, 2002). While this understanding the nuances of the underpinning theory is important, it is accord between what is predicted in theory and the empirical evidence that has been the focus of more practical debate.

Not surprisingly this has entailed exploring the more prominent of those techniques which have been used to estimate the VSL; namely, revealed preference methods (specifically hedonic wage studies) and stated preference methods. With regards to the former, Aldy and Viscusi (2007) find that for labour market data there is the inverted U-shaped relationship between age and VSL. However, the decline (in VSL) later in life is less pronounced (i.e. flatter) than the corresponding increase in earlier life. Krupnick (2007) reviews some of the evidence from stated preference studies and concludes that: this "… offers a mixed and somewhat confusing picture of whether a senior discount exists across the preferences in the United States and abroad" (p. 274). What is driving this ambiguity is itself unclear and may boil down to different handling of these analytical issues across the empirical record rather than reflecting preferences. OECD (2012) finds at best weak evidence for an inverted U-shaped relationship between VSL and mean age in their meta-sample.

A somewhat different approach is provided by Aldy and Smyth (2014). This makes use of a "simulated" experiment which involves examining the economic choices across the life cycle of (identical) "virtual" individuals (from the age of 20 years old) along with their implied WTP for mortality risk reductions. Each year (up to very old age), individuals in this simulated sample make work and leisure choices as well as consumption versus saving decisions, in response to economic circumstances and mortality risks. Those simulated individuals surviving to the next period are also "asked" in this exercise how much they would be willing to pay to reduce mortality risks in the coming year by a small amount. The results indicate a VSL which clearly varies over the life cycle of these virtual people exhibiting the inverted U-shaped relationship that many suspect exists. Given the aforementioned impasse in the stated preference literature, it remains to be seen whether this relationship works also for "real" people (i.e. outside the setting of labour market choices). Nevertheless, this simulated study provides possibly important clues. This notwithstanding, the prospect of constructing a robust and broadly agreed schedule of age-related VSL estimates remains elusive. However, one proposal for a straightforward procedure that does this is explored in Box 15.1.

Box 15.1. **The value of a (statistical) life-year**

The belief that age matters in computing VSL has led some to focus on simpler approaches; notably a "value of a statistical life-year" (or VSLY). Typically, the procedure here is to divide the VSL of someone of a given age, say 40, by the remaining years of life expectancy, say 40. Each "life year" would then be valued at: $VSLY = \frac{VSL_A}{T - A}$, where T is age at the end of a normal life and A is current age. In keeping with the lifetime consumption model, however, it is usually argued that the remaining life-years should themselves be discounted (by discount rate, r), so that the calculation becomes

$$VSLY = \frac{VSL_A}{\sum_t \frac{1}{(1+r)^{T-A}}}$$

As an example, someone aged 40, with a life expectancy of 78 and VSL of USD 5 million would have a VSLY of USD 131 579 on the simple approach, and USD 296 419 on the discounted

Box 15.1. **The value of a (statistical) life-year** *(cont.)*

approach.[a] While attractive in principle (because of its simplicity), securing a VSLY in this way from a VSL rests on substantial assumptions. First, as noted, the lifetime consumption model may not itself be capturing the features relevant to valuing remaining life years. Second, the resulting VSLY is very sensitive to the assumption made about the discount rate. Note that discount rates are not directly observed in this approach but are superimposed by the analyst.

In essence, what the VSLY approach does is to replace the assumption (implicit in the way that VSL is typically applied) that age does not matter with an alternative assumption that age not only matters but it matters in a particular way: i.e. as specified by the assumed VSLY conversion calculation such as a constant value that is discounted.[b] Proponents of the approach would argue that this has an intuitive appeal. That is, someone with, say, 40 years of life remaining and facing an immediate risk would tend to value "remaining life" more than someone with, say, 5 years of remaining life. Needless to say, there are counterarguments. For example, perhaps there is a scarcity value of time itself, i.e. fewer years left results in a higher WTP for the remaining years. And, of course, a natural question is whether the VSYL is justified by the empirical evidence about how WTP varies with age.

The discussion elsewhere in this chapter has reflected the view that while there appears to be a basis for an age/VSL relationship in some respects, the relationship is far from agreed especially in the context of the mixed results from stated preference evidence. However, there appears greater agreement that the schedule of life-year estimates in the typical VSLY approach does not appear to find support in practically any of the evidence to date. It is important not to interpret this as saying no age relationship exists. Rather it requires a more detailed search and in all likelihood will involve a more nuanced relationship than "mechanistic" straightline approaches assume. One starting point here might be, for example, Desaigues et al. (2011) which estimates more directly the VSLY using a stated preference approach across 9 European countries.[c]

a) The issue arises of whether individuals have already discounted the future when providing their WTP response if the approach used is a stated preference study. If so, the simple approach is relevant. If not, the discounted approach is more relevant.

b) The VSLY approach also assumes that one would value an additional life year the same at different ages. Hence, it does not permit, by assumption, a situation where a 40 year old person might not care very much if he or she was told that life expectancy would be 82 years instead of 83, but for someone aged 75 that difference *could* matter much more.

c) However, Alberinin (2017) states that "The Desaigues et al. study was conducted in nine countries for a total of only 1 463 respondents. Individuals were presented with a graph that clearly mislabels the change in life expectancy and the econometric analysis of the responses is well below acceptable standards".

15.6. Valuing morbidity

The previous sections have been concerned with the valuation of premature mortality. The reason for this is straightforward. Overwhelmingly, the evidence points to mortality as being by far the largest economic cost of health burdens related to the environment (see, for example, OECD, 2014, World Bank, 2016, and Cropper et al., 2010). Nevertheless, in environmental contexts, costs arising from morbidity, i.e. non-fatal ill health should not be ignored and are likely to significantly support the evidence for policy action when incorporated in a CBA. This was recognised in early work on the health costs of air pollution, including notably the ExternE study by the European Commission.

Morbidity endpoints even for air pollution are manifold and whether these valuations need to be and indeed should be carried out on each and every relationship between say air pollution and illness depends on a number of considerations. Hunt et al. (2016), for example, identify five key relationships for which they judge to be most promising. "Promising" on their judgement accords to whether the relationship is robust and (largely) unbiased and can be applied, as a practical matter, across OECD countries as well as The People's Republic of China and India. The end-points they identify are the following:

- Hospital admissions for both respiratory and cardiovascular problems related to ambient ozone or particulate matter (PM);
- Restricted activity days (RADs) or lost work days related to ambient ozone or PM;
- Chronic bronchitis in adults related to PM;
- Acute[11] bronchitis in children (aged 6 to 18 years) related to PM;
- Acute Lower Respiratory Illness (ALRI) in very young children (under 5 years old) related to PM.

The point here is that these are relationships for which there is quantitative evidence, in e.g. medical and epidemiological studies such as the estimation of dose-response functions, about the relation between exposure to a pollutant in e.g. urban air and physical health impact. Moreover based on an extensive literature review, Hunt et al. make recommendations about the unit values to attach these physical cases. These are illustrated in Table 15.5 and are intended to be base case values which might be adjusted according to income differences as discussed in Section 15.4.2.

Table 15.5. **Proposed unit values in for selected morbidity end-points**

USD, 2010 prices

Morbidity end-point	Central value	Range
Chronic bronchitis (per case)	334 750	41 700 to 889 000
Hospital admission (per case)	2 000	600 to 3 300
Work loss (per day)	Country specific	
RAD (per day)	170	41 to 268
Minor RADS (per day)	62	53 to 70
Acute bronchitis in children (per case)	464	301 to 511
ALRI in very young children (per case)	464	301 to 511

Source: Hunt et al. (2016).

The study by Cropper et al. (2010) provides an illustration of how a range of morbidity costs (along with mortality costs) might be calculated using the example of PM_{10} in Chinese cities. The authors consider a range of evidence about the physical link between air pollution exposure and a number of health end-points and conclude the following:

- For chronic bronchitis, the evidence for The People's Republic of China is that incidents increase by 4.8% per 10 µg per m^3 of PM_{10}. Put in terms of the dose-response functions discussed earlier in this Chapter (Section 15.2), this implies a coefficient β of 0.0048.
- For hospital admissions, there is a 0.7% and 1.2% change in number of incidents for respiratory and cardiovascular cases respectively as a result of 10 µg per m^3 of PM_{10}. Workdays lost as a result of each case is then calculated as the duration of a stay in hospital.

Table 15.6 reports the physical cases (panel a) and monetary costs (panel b) of exposure to PM_{10} in The People's Republic of China for 2003 as estimated in Cropper et al. The table makes clear the finding that mortality costs are the critical category of impact in

Table 15.6. **Health impacts and ambient air pollution in The People's Republic of China in 2003**

a) Health cases and incidents ('000s)

	Premature mortality	Morbidity			
		Chronic bronchitis	RHA	CHA	Workdays lost to hospital stays
Mean	394.0	305.3	223.6	216.3	9 210
Range	134.6 to 628.3	265.6 to 341.9	156.5 to 286.0	99.2 to 324.3	6 108 to 12 970

b) Health costs in billion Yuan

	Costs of premature mortality	Costs of morbidity			Total costs
		Chronic bronchitis	Direct hospital costs	Indirect hospital costs	
Mean	394.0	122.1	3.4	0.47	519.9
Range	135.6 to 641.1	106.2 to 137.7	1.9 to 4.8	0.26 to 0.67	243.9 to 783.3

Source: Cropper et al. (2010).

terms of economic burden. However, the morbidity value of chronic bronchitis accounts for almost a quarter of the total health costs. This should not be a surprise given the severity of this illness and the large number of estimated incidents in the study year.

15.7. Conclusions

While this Chapter has focused exclusively on analytical matters, it is important to remember that there are often vociferous and compelling criticisms of economists' "value of (statistical) life" estimates. These debates are important. Whether those criticisms mean putting health impacts beyond the reach of the cost-benefit analyst is debatable, although broadly the implications for CBA are similar to those discussed in Chapter 12, in relation to constraints on a CBA recommendation.

But it is useful to bear in mind that all decisions involving tolerance, acceptance or rejection of risk changes imply such valuations. The reason is very simple: risk reductions usually involve expenditure of resources, so that not spending those resources implies a sum of VSLs less than the resource cost. Conversely, spending the resources implies a sum of VSLs greater than the resource cost. Morrall (2003) provides a good illustration of this. Covering 76 regulations, Morrall derives implied VSLs ranging from USD 100 000 for a regulation covering childproof lighters, through USD 500 million for sewage sludge disposal regulations, and up to USD 100 billion for solid waste disposal facility criteria.[12]

Knowing these implied VSLs serve several purposes. First, as mentioned, they are a reminder that there is no "escape" from the valuation of life risks. Second, they serve as a measure of consistency across public agencies: the implied VSL for, say, transport risks should not be significantly different to the implied VSL for pollution reduction, unless there is a reason to suppose that the risks should be valued differently. Third, even if there is no consensus on "the" VSL, such exercises show that some policy measures are not credible in terms of their stated goal of cost-effectively saving (statistical) lives.

While knowing implicit VSL is useful, the progress in estimating *explicit* VSL suggests that CBA can do a lot more. Considerable strides have been made in recent years in terms of clarifying both the meaning and size of the VSL. One of the main issues has been how to "transfer" VSLs taken from e.g. non-environmental contexts to environmental contexts and country-to-country. The former consideration has given rise to concerns about nature of the risks involved.

Needless to say, this still requires that applications are done with care and judgement. In some areas, the literature offers firmer guidance here than in others. Notably, age may or may not be relevant in valuing immediate risks – the literature is arguably ambiguous and there is still a choice to be made between VSL and a life year approach, or perhaps using both although simpler approaches for the latter appear to lack firm empirical support. That said, in terms of practical guidelines, the empirical record has been important in translating findings in base or reference levels. Studies such as OECD (2012) have been important in distilling this empirical record into something highly usable as illustrated for the recent World Bank (2016) estimates of the global economic burden of air pollution.

Notes

1. This chapter is necessarily selective in its coverage since the literature on valuing human health impacts is now extremely large indeed. As a result, the focus here is only on a selection of the issues that have occupied attention in the recent literature.
2. This includes ambient pollution (exposure to particulate matter and ground level ozone) as well as indoor air pollution.
3. Terminology varies: VSL is also known as a "value of a prevented fatality" and, despite the warnings of economists about this phrase, "value of life". Cameron (2010) provides a compelling argument about the problems of all this terminology in communicating this economic concept and, in doing so, makes a case for a re-labelling around the more literal, but wordier, term: willingness to swap alternative goods and services for a micro-risk reduction in chance of sudden death.
4. These particles are defined in other ways depending on the size: TSP (total suspended particulates); PM_{10} (particles of 10 microns or less in diameter). There exist rough rules of thumb for converting between these units e.g.: $PM_{10}/0.55 = TSP$; $PM_{10}/2 = PM_{2.5}$. So for example, a 90 µg per m^3 reduction in TSP equates to a 50 µg per m^3 reduction in PM_{10}. A 50 µg per m^3 reduction in PM_{10} = 25 µg per m^3 reduction in $PM_{2.5}$.
5. More generally, these substantial impacts might also be examined via a general equilibrium approach, given that these changes are non-marginal and economy wide. See, for example, Marten and Newbold (2017) for a recent discussion of this.
6. Terminology can be confusing. The initial or baseline risk level needs to be distinguished from the change in the risk level brought about by the policy in question.
7. This is a weighted average where each mean VSL estimate in each individual study is weighted by the inverse of the number of observations from each SP survey.
8. There is also a separate issue of background risk. The expectation is that a "competing risk", i.e. some other risk to life independent of the risk being addressed by the policy measure, will reduce the WTP for the policy-related risk because of the "why bother" effect. That is, the competing risk reduces the chance that the individual will benefit from a reduction in the policy-related risk. But, in general, the effect will be very small. Eeckhoudt and Hammitt (2001) cite the example of a male worker aged about 40 in the USA. The mortality risk for that age group is 0.003 and this translates directly into a reduction in VSL of just 0.3 of a percentage point. However, risks of death from air pollution are highest for the elderly whose background risks are very high, i.e. they are at high risk of death from other causes. The "why bother" effect comes into play in a significant fashion. If the effect is significant, this would be expected to show up in expressed WTP for those who have high competing risks, notably (a) those who are in poor health anyway, and (b) the aged. The extent to which the empirical literature picks up this effect is discussed below with respect to health states and age.
9. In wage-risk studies any lack of a WTP-risk relationship may be due to "self-selection", where higher risk tolerant workers may be selecting the more hazardous employment. Meta-analyses of wage-risk studies also produce somewhat more mixed results. As risk increases, one would expect WTA (since what is measured is the premium on wages to accept higher risks) to vary directly with risk levels. On the other hand, the self-selection effect may mean that less risk-averse workers gravitate to higher risk occupations. Mrozek and Taylor (2002) find both effects, i.e. a rising WTA at first followed

by a reduction thereafter. This "risk loving" effect has been noted in other occupational studies as well (for a summary, see Hammitt, 2002). On the other hand, Viscusi (2004) finds that wage premia vary directly with death risks and with injury risks in occupations, and Viscusi and Aldy's (2003) meta-analysis of wage risk studies for the USA finds VSL that vary directly with risk reduction but with only a minor effect of high risk on VSL (the implied VSL is USD 12-22 million for low risks and a tenfold increase in risks changes this to USD 10-18 million).

10. Roy and Braathen (2017) uses a slightly different formula:

    $VSL\ C_{2015} = VSL\ OECD_{2010} \times (Y\ C_{2010}/Y\ OECD_{2010})^{\beta} \times (1 + \%\Delta P + \%\Delta Y)^{\beta}$, taking into account the changes in price and income levels since the baseyear used. One could argue for an additional modification: $VSL\ C_{2015} = VSL\ OECD_{2010} \times (Y\ C_{2010}/Y\ OECD_{2010})^{\beta} \times (1 + \%\Delta P) \times (1 + \%\Delta Y)^{\beta}$, thus not adjusting the change in the price level for the income elasticity.

11. Defined as an incident within the last 12 months.

12. Taking a USD 7 million "cut-off" point, Morrall finds that nearly all regulations aimed at safety pass a cost-benefit test, but less than 20% of regulations aimed at reducing cancers pass such a test. Finally, by employing "risk-risk" or "health-health" analysis (see Chapter 17), Morrall shows that USD 21 million of public expenditure gives rise to one statistical death. All policies cost money which ultimately comes from taxation, reducing the disposable income of taxpayers. Some of that forgone income could have been spent on life-saving measures. Hence, all government expenditure might cause life loss, at least to some extent. Any measure that implies a VSL of more than USD 21 million "does more harm than good", i.e. it generates more deaths than lives saved. 27 of the 76 regulations studies fail this test. Comparable studies exist for Sweden where the cut-off point is USD 6.8-9.8 million – see Gerdtham and Johannesson (2002) and the United Kingdom, where the cut-off is around USD 8 million (Whitehurst, 1999).

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ANNEX 15.A1

Deriving the value of a statistical life

The standard approach to the VSL is to assume that an individual's utility function for wealth (W) and mortality risk (p) is expressed in the utility function:

$$U(p, W) = (1 - p) . u_a(W) + p . u_d(W) \quad [15.A1.1]$$

where U is (expected) utility, $u_a(W)$ is the utility conditional on surviving – i.e. the utility of being alive – and $u_d(W)$ is the utility conditional on dying. It is assumed that $u_a' > 0$ and $u_a'' < 0$. The former assumption says that marginal utility of wealth is increasing in wealth, and the second says that the individual is averse to gambles with expected value of zero, i.e. individuals are averse to financial risk.

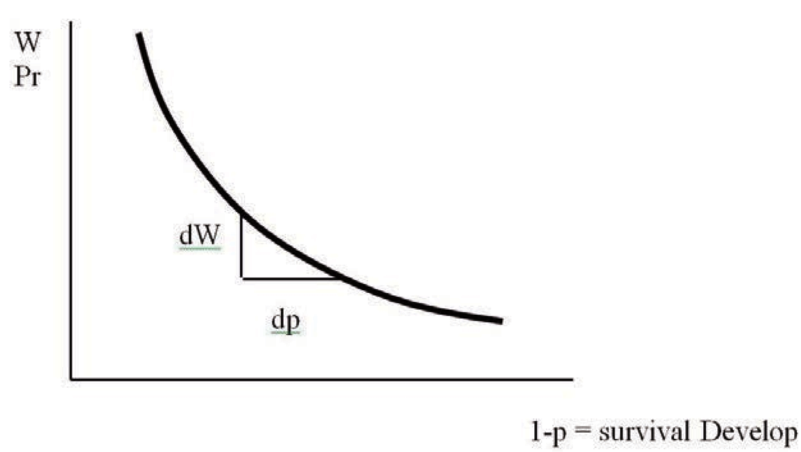
This is a one-period model, and for the sake of simplicity, u_d (W) can be interpreted as including bequests etc. so that it is not necessarily equal to zero, i.e. $u_d' \geq 0$. It is further assumed that:

$$u_a(W) > u_d(W) \quad \text{and}$$

$$u_a'(W) > u_d'(W) \quad [15.A1.2]$$

The second condition simply means that more wealth provides more utility if the individual survives than if he/she dies. Put another way, additional wealth yields more utility in life than as a bequest.

The corresponding indifference curve is:



Differentiating [15.A1.1] whilst holding utility constant gives:

$$\frac{dU(.)}{dp} = (1-p)\cdot u_a'(W) - u_a(W) + p\cdot u_d'(W) + u_d(W) = 0 \qquad [15.A1.3]$$

so that

$$VSL = \frac{dW}{dp}\Big|_{EU=\ constant} = \frac{u_a(W) - u_d(W)}{(1-p).u_a'(W) + p.u_d'(W)} \qquad [15.A1.4]$$

The numerator shows the difference between utility if the individual survives or dies in the current period. The denominator is the expected marginal utility of wealth, conditional on survival and dying, each event being weighted by the relevant probabilities. The denominator is often called the "expected utility cost of funds" or the "expected utility cost of spending".

Baseline risk

Given the inequalities [15.A1.2], VSL > 0. VSL also increases with baseline risk, p, the so-called "dead anyway" effect (Pratt and Zeckhauser, 1996). Hammitt (2000) points out that this effect cannot be large for small risk changes because survival probabilities for any year are much higher than mortality probabilities ((1-p) is large, p is small). As p increases, the numerator in [15.A1.4] is unchanged because p does not affect it. But the denominator changes since the first expression declines and the second increases. Given the likely probabilities, the decline outweighs the increase and the denominator thus decreases. VSL rises with baseline risk, but not by much.

Wealth

The effect of wealth changes on VSL depends on financial risk aversion in the two states – survival and death. Risk neutrality and risk aversion are sufficient to ensure that VSL rises with W. Since $u_a'(W) > u_d'(W)$ the numerator increases in wealth. Since $u_a''(W) < 0, u_d''(W) < 0$, the denominator declines with wealth. Hence VSL rises with wealth.

Health status

The relationship between VSL and health status on survival is strictly indeterminate, although many studies assume that VSL will be higher for survival in good health than for survival in poor health, which seems intuitively correct. Hammitt (2000) points out that survival in bad health may limit the individual's ability to increase utility by spending money – the marginal utility of wealth may be lower for survival in bad health than in good health. The denominator in [15.A1.4] is smaller if survival means bad health. But the numerator is also smaller, so the relationship between VSL and health state is dependent on exact values and could be positive or negative.

Latency

Equation [15.A1.4] says nothing about latency, i.e. exposure to risks now may result in death much later (e.g. arsenicosis, asbestosis etc.). The relevant VSL (call it VSL_{lat}) is:

$$VSL_{lat} = \frac{VSL_T}{(1+s)^T} . P_T \qquad [15.A1.5]$$

where VSL_{lat} is the VSL now for an exposure risk occurring now, T is the latency period after which the individual dies, s is the discount rate (technically, the individual's discount rate) and P_T is the probability that the individual will survive the latency period, i.e. the probability that he/she does not die from other causes in the interim period. Essentially, then, the relevant VSL is the discounted value of the future VSL at the time the risk effect occurs, adjusted for the probability of surviving during the latency period. If WTP varies with income and income increases with time, then, rather than discounting future WTP at the relevant discount rate, a *net* discount rate may be used. If *s* is the discount rate and WTP grows as n % per annum, the net discount rate will be (s-n) % per annum. A convenient case occurs where s = n since this reduces the problem to one of using undiscounted values.

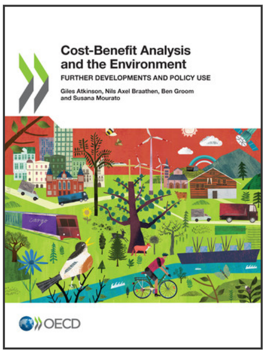
Hammitt and Liu (2004) present a somewhat more sophisticated version of [15.A1.5] for a latent effect where the risk change occurs as a "blip', i.e. a temporary risk reduction as opposed to a permanent reduction of risk. (For a permanent risk reduction, WTP needs to be summed for each of the future periods). Their equation is:

$$WTP_0 = \frac{WTP_T}{\left(1/(1+s)\right)^T .a_T.(1+g)^{T\eta}} \qquad [15.A1.6]$$

where WTP_0 is willingness to pay for a risk reduction now, WTP_T is willingness to pay for a risk reduction in T years' time, s is the personal discount rate, a is a factor linking age to WTP (a = 1 if age has no effect on willingness to pay, with a < 1 being the usual expectation), g is the growth rate of income and η is the income elasticity of willingness to pay for risk reduction. Equation [15.A1.6] thus makes an explicit attempt to modify the VSL equation for (a) age and (b) interim income growth during a latency period.

Age

Equation [15.A1.4] does not tell us if WTP (and hence VSL) varies with age. Age is usually thought to have two potentially offsetting effects: (a) the older one gets, the fewer years are left so the benefit of any current reduction in risk declines – we would expect VSL to decline with age; (b) the opportunity cost of spending money on risk reduction declines as time goes by because savings accumulate, so WTP for risk reduction may actually rise with age. Technically, therefore, VSL may vary with age in an indeterminate manner.



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