

estimate to each fatal case of bladder cancer in the benefits model.

A review of the available literature did not reveal any studies that specifically measured the WTP to avoid risks of contracting nonfatal cases of bladder cancer. Instead, two alternates were used, the WTP to avoid the risk of contracting a case of curable lymph cancer (lymphoma) and the WTP to avoid a case of chronic bronchitis. The SAB suggested this approach in their review of the Arsenic Rule (USEPA 2001e). The median risk-risk trade-off for a curable case of lymphoma was equivalent to 58.3 percent of the risk

attributed to reducing the chances of facing a sudden death and are derived from the Magat *et al.* study (1996). Therefore, the Agency applies the 58.3 percent to the VSL distribution to derive a range of value for non-fatal cancers with a mean WTP value of \$2.8 million (\$4.8 million * 58.3 percent) at a 1990 price level. The WTP for avoiding a case of chronic bronchitis is based on the same methodology used for the Stage 1 DBPR (see Stage 2 DBPR EA (USEPA 2003i) for a complete discussion). The estimate is based on a lognormal distribution that uses the risk-dollar

tradeoff estimate and has a mean of \$587,500, standard deviation of \$264,826, and a maximum value of \$1.5 million at 1998 price values.

Update factors. All valuation parameters must be updated to the same price level so comparisons can be made in real terms. Values for VSL, WTP, and the morbidity increment used in the model are updated based on adjustment factors derived from Bureau of Labor Statistics (BLS) consumer price index (CPI) data so that each represents a year 2000 price level. Table VII-4 summarizes these updates.

Table VII-4. VSL, WTP, and Morbidity Increment Price Level Updates

Valuation Parameter	Base Year	Mean Value in Base Year (Millions)	CPI Update Factor	Values at Year 2000 Price Level (Millions)			
				Mean	Median	Lower (5th %tile)	Upper (95th %tile)
Morbidity Increment	1996	\$ 0.1	1.14	\$ 0.1	N/A	N/A	N/A
VSL	1990	\$ 4.8	1.32	\$ 6.3	\$ 5.5	\$ 1.0	\$ 14.5
WTP - Non-Fatal Lymphoma	1990	\$ 2.8	1.32	\$ 3.7	\$ 3.2	\$ 0.6	\$ 8.5
WTP - Chronic Bronchitis	1998	\$ 0.6	1.06	\$ 0.6	\$ 0.6	\$ 0.3	\$ 1.1

Notes: Morbidity increment value is presented as a point estimate.

Source: Economic Analysis (USEPA 2003i) Exhibit 5.29

Although the price level (year 2000) is held constant throughout the benefits model, projections of benefits in future years are subject to income elasticity adjustments. Income elasticity adjustments represent changes in valuation in relation to changes in real income. For fatal cancers, the Agency used a triangular distribution with a central estimate of 0.40 (low end: 0.08; high end: 1.00) to represent the uncertainty of the income elasticity value. For non-fatal cancers, the Agency uses a triangular distribution with a central estimate of 0.45 (low end: 0.25; high end: 0.60). These distributions are used as assumptions in the Monte Carlo simulation to further characterize uncertainty in benefits estimates.

In order to apply the income elasticity values in the model, they are combined with projections of real income growth over the time frame for analysis. Population and real gross domestic product (GDP) projections are combined to calculate per-capita real GDP values. A more detailed discussion of these adjustments is in Chapter 5 of the EA (USEPA 2003i).

The development of cancer due to exposure to environmental carcinogens involves a complex set of processes that are not well-understood for most specific substances. In general, however, the development of cancer involves some time period, usually referred to as the latency period, between the initial exposure and the manifestation of disease. Defining a latency period is highly uncertain because the mode of action for most chemical contaminants are poorly understood. Latency periods in humans often involve many years, even decades.

EPA recognizes that despite uncertainties in the latency period associated with different types of carcinogens, it is unlikely that all cancer reduction benefits would be realized immediately upon exposure reduction. If it is assumed that lower risk is attained immediately upon reduction in exposure, this would tend to overestimate the benefits. On the other hand, assuming that no risk reduction occurs for some period of time following exposure reduction may lead to an underestimation of the benefits. There will likely be some transition period as

individual risks become more reflective of the new lower exposures than the past higher exposures.

Recently, the Arsenic Rule Benefits Review Panel of the EPA Science Advisory Board (SAB) addressed this issue in detail and provided some guidance for computing benefits to account for this transition period between higher and lower steady-state risks (USEPA 2003s). The Arsenic Rule Benefits Review Panel coined the term "cessation-lag" to emphasize the focus on the timing of the attenuation of risk after reduction in exposures to avoid confusion with the more traditional term of "latency" that reflects the increased risk⁴ from the time of initial exposure.

⁴ SAB included the following in its report on arsenic to emphasize this difference: "An important point is that the time to benefits from reducing arsenic in drinking water may not equal the estimated time since first exposure to an adverse effect. A good example is cigarette smoking: the latency between initiation of exposure and an increase in lung cancer risk is approximately 20 years. However, after cessation of exposure, risk for lung cancer begins to decline rather quickly. A benefits analysis of smoking cessation programs

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