Appendix A:
Supplement to the Economic Assessment

SUPPLEMENT TO THE ECONOMIC ASSESSMENT

There are many ways that increasing the size of New Jersey's renewable energy portfolio can affect the environment and the economy. One partial equilibrium outcome is that the unadjusted price of electrical energy will increase. This is because conventional sources have been selected by the market economy since they are perceived to be less expensive than alternative fuels, at least given the current electricity generation set in place. On the other hand, a point of enhancing the renewable portfolio is to meet environmental objectives. Thus, the price increase of electricity is a prime component of the costs for meeting those of objectives. Such impacts are discussed elsewhere in detail using an economic forecast as the modeling vehicle. But there are other potential costs and benefits of converting to electricity generation based on renewable energy sources. Indeed, while the relative costs of expanding investment in a renewable portfolio is embedded in average electricity prices, the relative benefits of the investments are not counted.

The benefits of a specific investment are typically measured in terms of the jobs and wealth that are created in the wake of that investment. Increases in these measures occur due to the augmentation of generation technology production, the installation of the plant, and the operation and maintenance of the facility. Naturally, to identify the changes of these measures due to conversion to a new technology, the benefits that would accrue to the new technology must be compared to the set of benefits that would have accrued had no policy change taken place. We assumed here, as throughout this report, that natural gas would serve as the prime conventional energy resource and that gas turbines would be used to generate electricity. Moreover, we presumed that conventional sources constructed in the future would be located out of state.

The net investment benefits of two renewable fuel technologies—landfill biogas and biomass—were acknowledged as negligible. Electricity produced from landfill biogas was perceived at best to maintain its current low-level production and perhaps even decline through the end of the study period. Gasified and fluidized-bed biomass technologies were recognized to be insufficiently price-competitive by 2020 (Navigant, 2004, p. 235): hence, they are supposed to make insignificant into New Jersey's energy supplies through 2020. While direct biomass conversion is presently limited, the technology being used is, in any case, largely conventional. Moreover, while policies could be developed to induce the location of such facilities in New Jersey, those same policies could serve to subsidize their eventual conversion to conventional power facilities. Hence, the economic benefits of both biomass technologies were not investigated.

The two remaining renewable energy sources for which economic benefits are considered are (1) solar photovoltaics and (2) wind. The remainder of this section discusses how their economic benefits were estimated.

In order to estimate the benefits to the New Jersey economy of these two technologies, the engineering costs of the manufacture and installation of the two

technologies had to be identified. Those for solar photovoltaics are displayed in Table A.1. It was presumed that the cost structure for photovoltaics systems connected with commercial, industrial, and civic/institutional buildings would not be much different. Engineering cost estimates for a wind park are shown in Table A.2.

Table A.1: Cost Breakdown of a Typical 8 KW Residential Solar Photovoltaic Array

Material/Service	Cost in 2004 Dollars
Marketing and Sales	3,200
Engineering and Design	480
Post-installation Servicing	960
General and Administrative	2,400
Modules	29,280
Inverters	7,680
Mounting	3,360
Miscellaneous Material	1,920
Array Labor	5,360
Electrician Labor	3,680
Total	58,320

Source: Lyle Rawlings, Advanced Solar Products, Hopewell, New Jersey.

Table A.2: An Example Cost Analysis for a 60 MW Wind Park

Item	Cost in 2004 Dollars
40 1.5 MW turbines	46,640,000
Site preparation & grid connection	9,148,000
Interest and contingencies	3,514,000
Project development & feasibility study	965,000
Engineering	611,000
Total	60,878,000

Source: Masters (2004, Table 6.8, p. 373).

With only minor modification to align the materials, services and other items so they were aligned with model sectors, the data in Tables A.1 and A.2 were entered into the R/Econ I-O model for the State of New Jersey.

For each of the two technologies, two different R/Econ I-O model runs were undertaken. In the case of PV, we first assumed that the technology was only installed in New Jersey. Prior experience has revealed that on average 90 percent of all contracting

work in New Jersey is performed by contractors who live in the state. Hence, we applied this proportion in the present study. Table A.3 shows a summary of the results on the economic and tax impacts on New Jersey of 8 Mw of PV installation in the State. Note that the direct output effects (the first column of II.1 in Table A.3) are not 1,000 times the total spending in Table A.1. This is because the PV units are assumed to be produced in New Jersey only in so much as they would normally meet state-based PV demand. Needless to say, a substantial portion of spending on solar photovoltaic units currently tends to take place outside of the State. Indeed, based on the results shown in Table A.3, only about a third ($19 million) of the $58 million initial investment is assumed to be spent on goods and services within the State under current circumstances, and more than half of this is for construction services required to install the arrays in place within the State. Moreover, looking at the bottom of Table A.3, it is clear that $1 million of 2002 dollars spent on installed PV will yield 3.8 full-time-equivalent jobs, about $280,000 in total state wealth (gross state product), $210,000 in earnings to state workers, and a total of $19,000 split between state and local tax revenues. Generally speaking, these are less-than-stellar returns to a state-based infrastructure investment. On the other hand, the average annual earnings per job are estimated to be $54,700, 15% above the average for the state ($47,420 in 2002). Of course, environmental savings of PV compared to conventional fuels are not included in these calculations.

The second run for PV assumes a policy that invokes a mandate that all PV installed in New Jersey is also produced in the State. Hence, not only were 90 percent of contractors New Jerseyans, but all design, marketing, sales, and production aspects of the PV installed in New Jersey buildings also are presumed to take place in the State. Table A.4 summarizes the economic impacts on the State of bring this policy into play. In general, the effects of this New Jersey-only technology policy yields an economic impact that is nearly two and a half times larger than if no such policy was put in place. In this case, each million spent on PV installations will produce about 8.7 jobs, $700,000 in wealth, $516,000 in earnings, and $55,000 in state and local taxes. While these impacts are low in terms of jobs, they comport well otherwise with other state-based infrastructure investments. Moreover, the earnings per jobs are estimated to be even higher than in the base case—at $59,600, 10% higher…or 25% above the state's annual average in 2002.

In the case of wind power, the basic assumption was that wind power demand would be met from sources outside of New Jersey but within the PJM market. Hence, increasing wind's share of the RPS was understood to have negligible economic impacts upon the state without prompting the industry with incentives. The other alternative is to apply policies that not only induce the installation of wind-based electric power plants within the State in the form of offshore turbines, but also to induce the production of the turbines and towers themselves. The economic impacts of this recourse are displayed in Table A.5. In general, the economic impacts per $1 million of installing and producing wind power are just slightly lower than those for PV produced in the State: 7.6 jobs, $636,000 in wealth, $462,000 in earnings, and $53,000 in state and local taxes. Due to its larger yield in manufacturing jobs, it impacts include jobs with average annual earnings of $60,600, slightly above those for State-based PV.

Table A.3: Economic and Tax Impacts on New Jersey of Installing 8 MW of Residential Photovoltaic Systems (Year 2000 Dollars)

	Economic Component			
	Output ($000)	Employment (jobs)	Income ($000)	Gross State Product ($000)
I. TOTAL EFFECTS (Direct and Indirect/Induced)*				
1. Agriculture	24.9	0	2.6	4.9
2. Agri. Serv., Forestry, & Fish	11.6	0	5.7	9.6
3. Mining	6.5	0	2.3	4.3
4. Construction	10,243.8	106	6,504.4	8,482.6
5. Manufacturing	5,553.9	25	1,467.9	1,776.0
6. Transport. & Public Utilities	1,034.3	4	263.8	415.0
7. Wholesale	693.2	4	281.9	297.7
8. Retail Trade	1,550.1	25	581.2	907.0
9. Finance, Ins., & Real Estate	1,664.8	9	554.6	1,139.8
10. Services	6,726.6	49	2,451.4	3,338.4
Private Subtotal	27,509.7	222	12,115.9	16,375.3
Public				
11. Government	58.5	0	17.9	28.5
Total Effects (Private and Public)	27,568.3	222	12,133.8	16,403.7
II. DISTRIBUTION OF EFFECTS/MULTIPLIER				
1. Direct Effects	19,113.5	156	9,326.1	12,246.5
2. Indirect and Induced Effects	8,454.8	66	2,807.6	4,157.3
3. Total Effects	27,568.3	222	12,133.8	16,403.7
4. Multipliers (3/1)	1.442	1.427	1.301	1.339
III. COMPOSITION OF GROSS STATE PRODUCT				
1. Wages--Net of Taxes				11,230.0
2. Taxes				2,204.7
a. Local				276.3
b. State				252.9
c. Federal				1,675.5
General				374.6
Social Security				1,300.9
3. Profits, dividends, rents, and other				2,969.0
4. Total Gross State Product (1+2+3)				16,403.7
IV. TAX ACCOUNTS				
		Business	**Household**	**Total**
1. Income --Net of Taxes		11,230.0	0.0	
2. Taxes		2,204.7	2,462.1	4,666.8
a. Local		276.3	315.6	591.9
b. State		252.9	276.3	529.2
c. Federal		1,675.5	1,870.2	3,545.7
General		374.6	1,870.2	2,244.8
Social Security		1,300.9	0.0	1,300.9
EFFECTS PER MILLION DOLLARS OF INITIAL EXPENDITURE				
Employment (Jobs)				3.8
Income				208,055.4
State Taxes				9,073.4
Local Taxes				10,149.5
Gross State Product				281,270.8
INITIAL EXPENDITURE IN DOLLARS				58,320,000.0

Note: Detail may not sum to totals due to rounding.

*Terms:

Direct Effects --the proportion of direct spending on goods and services produced in the specified region.
Indirect Effects--the value of goods and services needed to support the provision of those direct economic effects.
Induced Effects--the value of goods and services needed by households that provide the direct and indirect labor.

Table A.4: Economic and Tax Impacts on New Jersey of Installing 8 MW of Residential Photovoltaic Systems, Assuming All Production Takes Place in the State (Year 2000 Dollars)

	Economic Component			
	Output (000$)	Employment (jobs)	Income (000$)	Gross State Product (000$)
I. TOTAL EFFECTS (Direct and Indirect/Induced)*				
1. Agriculture	60.4	0	6.4	11.7
2. Agri. Serv., Forestry, & Fish	47.9	1	24.7	40.2
3. Mining	9.4	0	3.2	6.1
4. Construction	11,173.3	108	6,632.8	8,792.5
5. Manufacturing	46,388.8	193	14,318.2	18,542.0
6. Transport. & Public Utilities	2,960.6	11	724.8	1,180.8
7. Wholesale	2,645.1	15	1,075.6	1,136.1
8. Retail Trade	3,767.7	61	1,408.6	2,192.6
9. Finance, Ins., & Real Estate	4,375.3	22	1,427.9	3,005.2
10. Services	11,454.2	93	4,432.3	5,691.5
Private Subtotal	82,882.7	504	30,054.6	40,598.7
Public				
11. Government	180.1	1	55.3	89.3
Total Effects (Private and Public)	83,062.9	505	30,109.9	40,687.9
II. DISTRIBUTION OF EFFECTS/MULTIPLIER				
1. Direct Effects	58,320.0	319	21,894.6	28,733.6
2. Indirect and Induced Effects	24,742.9	186	8,215.3	11,954.3
3. Total Effects	83,062.9	505	30,109.9	40,687.9
4. Multipliers (3/1)	1.424	1.582	1.375	1.416
III. COMPOSITION OF GROSS STATE PRODUCT				
1. Wages--Net of Taxes				27,650.1
2. Taxes				5,688.1
a. Local				943.7
b. State				787.3
c. Federal				3,957.1
General				728.9
Social Security				3,228.3
3. Profits, dividends, rents, and other				7,349.8
4. Total Gross State Product (1+2+3)				40,687.9
IV. TAX ACCOUNTS				
		Business	**Household**	**Total**
1. Income --Net of Taxes		27,650.1	0.0	
2. Taxes		5,688.1	6,109.6	11,797.6
a. Local		943.7	783.1	1,726.8
b. State		787.3	685.6	1,472.9
c. Federal		3,957.1	4,640.8	8,598.0
General		728.9	4,640.8	5,369.7
Social Security		3,228.3	0.0	3,228.3
EFFECTS PER MILLION DOLLARS OF INITIAL EXPENDITURE				
Employment (Jobs)				8.7
Income				516,287.6
State Taxes				25,254.9
Local Taxes				29,609.3
Gross State Product				697,666.5
INITIAL EXPENDITURE IN DOLLARS				58,320,000.0

Note: Detail may not sum to totals due to rounding.

*Terms:

Direct Effects --the proportion of direct spending on goods and services produced in the specified region.

Indirect Effects--the value of goods and services needed to support the provision of those direct economic effects.

Induced Effects--the value of goods and services needed by households that provide the direct and indirect labor.

Table A.5: Economic and Tax Impacts on New Jersey of Installing 60 MW Off-shore Wind Park, Assuming All Production Takes Place in the State (Year 2000 Dollars)

	Economic Component			
	Output (000$)	Employment (jobs)	Income (000$)	Gross State Product (000$)
I. TOTAL EFFECTS (Direct and Indirect/Induced)*				
1. Agriculture	60.2	0	6.2	11.5
2. Agri. Serv., Forestry, & Fish	46.7	1	24.1	39.2
3. Mining	108.3	1	37.3	71.1
4. Construction	6,601.3	40	3,066.3	4,340.5
5. Manufacturing	56,821.3	240	16,624.7	21,836.4
6. Transport. & Public Utilities	7,360.5	21	1,457.5	2,812.5
7. Wholesale	2,999.4	17	1,219.7	1,288.3
8. Retail Trade	3,704.1	60	1,382.6	2,150.1
9. Finance, Ins., & Real Estate	4,240.8	22	1,420.2	2,854.4
10. Services	6,510.1	62	2,838.8	3,233.3
Private Subtotal	88,452.7	463	28,077.5	38,637.1
Public				
11. Government	218.5	1	66.6	105.3
Total Effects (Private and Public)	88,671.2	464	28,144.0	38,742.4
II. DISTRIBUTION OF EFFECTS/MULTIPLIER				
1. Direct Effects	60,878.0	262	18,727.9	25,558.5
2. Indirect and Induced Effects	27,793.2	203	9,416.1	13,183.9
3. Total Effects	88,671.2	464	28,144.0	38,742.4
4. Multipliers (3/1)	1.457	1.775	1.503	1.516
III. COMPOSITION OF GROSS STATE PRODUCT				
1. Wages--Net of Taxes				25,637.1
2. Taxes				5,524.4
a. Local				1,033.9
b. State				841.2
c. Federal				3,649.3
General				631.8
Social Security				3,017.5
3. Profits, dividends, rents, and other				7,580.9
4. Total Gross State Product (1+2+3)				38,742.4
IV. TAX ACCOUNTS				
		Business	**Household**	**Total**
1. Income --Net of Taxes		25,637.1	0.0	
2. Taxes		5,524.4	5,710.7	11,235.1
a. Local		1,033.9	732.0	1,765.9
b. State		841.2	640.8	1,482.1
c. Federal		3,649.3	4,337.8	7,987.1
General		631.8	4,337.8	4,969.6
Social Security		3,017.5	0.0	3,017.5
EFFECTS PER MILLION DOLLARS OF INITIAL EXPENDITURE				
Employment (Jobs)				7.6
Income				462,302.2
State Taxes				24,344.8
Local Taxes				29,007.5
Gross State Product				636,394.0
INITIAL EXPENDITURE IN DOLLARS				60,878,000

Note: Detail may not sum to totals due to rounding.

*Terms:

Direct Effects --the proportion of direct spending on goods and services produced in the specified region.

Indirect Effects--the value of goods and services needed to support the provision of those direct economic effects.

Induced Effects--the value of goods and services needed by households that provide the direct and indirect labor.

This part of Appendix A includes the BASELINE forecast used in this study as well as 4 other scenarios discussed in Chapter 2.

Table A.6: R/ECON BPU BASELINE FORECAST

Electric Utilities: Prices, Usage, Taxes, etc.	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	Growth 2004 - 2020
Electric Price per Kilowatt Hour	$0.100	$0.100	$0.100	$0.100	$0.101	$0.102	$0.102	$0.103	$0.103	$0.104	$0.105	$0.105	$0.106	$0.106	$0.107	$0.108	$0.108	8.0%
Residential	$0.111	$0.111	$0.110	$0.110	$0.110	$0.111	$0.111	$0.112	$0.112	$0.113	$0.113	$0.114	$0.115	$0.115	$0.116	$0.117	$0.117	5.4%
Other	$0.093	$0.095	$0.095	$0.095	$0.097	$0.097	$0.098	$0.098	$0.099	$0.099	$0.100	$0.100	$0.101	$0.102	$0.102	$0.103	$0.104	11.8%
Electricity Usage in 1000 Megawatt Hours	66,537	73,644	75,482	76,615	77,620	78,629	79,635	80,621	81,636	82,679	83,794	84,952	86,064	87,151	88,242	89,368	90,539	36.1%
Residential	26,367	26,538	26,946	27,405	27,864	28,315	28,758	29,195	29,632	30,073	30,522	30,982	31,450	31,923	32,397	32,872	33,350	26.5%
Other	40,170	47,106	48,536	49,210	49,756	50,314	50,877	51,425	52,004	52,607	53,271	53,970	54,614	55,228	55,845	56,496	57,189	42.4%
Electric Revenues ($ Billions)	$6.7	$7.4	$7.6	$7.7	$7.9	$8.0	$8.2	$8.3	$8.5	$8.6	$8.8	$8.9	$9.1	$9.3	$9.5	$9.7	$9.8	47.8%
Residential	$2.9	$2.9	$3.0	$3.0	$3.1	$3.1	$3.2	$3.3	$3.3	$3.4	$3.4	$3.5	$3.6	$3.7	$3.8	$3.8	$3.9	33.3%
Other	$3.7	$4.5	$4.6	$4.7	$4.8	$4.9	$5.0	$5.0	$5.1	$5.2	$5.3	$5.4	$5.5	$5.6	$5.7	$5.8	$5.9	59.2%
Energy Taxes ($ Millions)	$1,108.3	$932.6	$897.7	$856.7	$869.4	$881.4	$893.8	$907.2	$922.2	$938.4	$955.6	$973.5	$991.3	$1,008.5	$1,025.4	$1,042.5	$1,060.3	-4.3%
Energy Taxes - TEFA	$858.8	$822.6	$842.7	$856.7	$869.4	$881.4	$893.8	$907.2	$922.2	$938.4	$955.6	$973.5	$991.3	$1,008.5	$1,025.4	$1,042.5	$1,060.3	23.5%
Sales	$697.1	$630.1	$639.4	$647.8	$658.5	$667.4	$675.7	$684.4	$693.6	$703.0	$713.2	$723.7	$734.1	$744.5	$755.1	$766.1	$777.6	11.5%
Corporate Business	$161.7	$192.6	$203.3	$208.9	$210.9	$214.0	$218.1	$222.9	$228.6	$235.4	$242.5	$249.8	$257.2	$264.0	$270.3	$276.4	$282.7	74.8%
Transitional Facility Assessment	$249.5	$110.0	$55.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	
Gross State Product for Utilities ($ Billions)	$8.04	$8.16	$8.40	$8.52	$8.56	$8.63	$8.72	$8.82	$8.95	$9.09	$9.25	$9.41	$9.57	$9.72	$9.86	$9.99	$10.12	26.0%
Gross State Product ($Billions 2000=100)	$354.57	$364.10	$373.83	$383.97	$393.62	$403.29	$414.16	$426.09	$438.96	$453.03	$468.20	$484.17	$500.86	$518.30	$536.48	$555.63	$575.88	62.4%
Employment at Utilities (Thousands)	15.4	15.3	15.4	15.4	15.4	15.3	15.3	15.3	15.3	15.3	15.2	15.2	15.2	15.2	15.2	15.2	15.2	-1.2%
Electric Utilities	8.9	8.8	8.7	8.6	8.6	8.5	8.5	8.4	8.4	8.4	8.4	8.3	8.3	8.3	8.3	8.3	8.3	-6.4%
Other	6.4	6.6	6.7	6.8	6.8	6.8	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	7.7%
NonAgricultural Employment (Thousands)	4030.2	4079.7	4133.8	4183.6	4227.1	4270.3	4317.4	4363.2	4411.9	4463.4	4521.9	4576.9	4628.1	4677.9	4730.5	4784.8	4845.0	20.2%
NJ Consumer Price Index (1982-84=100)[1]	196.6	199.4	201.8	205.3	209.5	213.9	218.7	224.2	229.7	235.3	241.1	247.1	253.2	259.4	265.8	272.4	279.1	41.9%
Renewable Portfolio Standard	3.25	3.40	4.13	5.12	6.21	6.50	6.50	6.50	6.50	6.50	6.50	6.50	6.50	6.50	6.50	6.50	6.50	100.0%
Class 1: Photovoltaics, et al.	0.75	0.90	1.63	2.62	3.71	4.00	4.00	4.00	4.00	4.00	4.00	4.00	4.00	4.00	4.00	4.00	4.00	433.3%
Class 2:Hydroelectric and Waste-to-Energy	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	0.0%

1 The NJ CPI is a population-weighted average of the CPIs for NY-NJ-CT and PA-NJ.

Table A.7: R/ECON BPU STRAIGHTLINE RPS INCREASE
2009 to 20% in 2020

Electric Utilities: Prices, Usage, Taxes, etc.	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020
Electric Price per Kilowatt Hour	$0.100	$0.100	$0.100	$0.100	$0.101	$0.103	$0.105	$0.107	$0.110	$0.113	$0.116	$0.119	$0.122	$0.125	$0.128	$0.131	$0.134
Residential	$0.111	$0.111	$0.110	$0.110	$0.110	$0.111	$0.112	$0.114	$0.117	$0.119	$0.122	$0.125	$0.127	$0.130	$0.133	$0.136	$0.138
Other	$0.093	$0.095	$0.095	$0.095	$0.097	$0.098	$0.100	$0.103	$0.106	$0.109	$0.112	$0.115	$0.119	$0.122	$0.125	$0.129	$0.131
Electricity Usage in 1000 Megawatt Hours	66,536	73,643	75,480	76,614	77,619	78,552	79,516	80,484	81,489	82,527	83,639	84,797	85,909	86,996	88,089	89,216	90,444
Residential	26,367	26,538	26,946	27,405	27,863	28,308	28,744	29,177	29,610	30,049	30,497	30,956	31,423	31,895	32,369	32,844	33,327
Other	40,169	47,105	48,535	49,209	49,755	50,244	50,772	51,307	51,879	52,479	53,142	53,841	54,486	55,101	55,720	56,371	57,117
Electric Revenues ($ Billions)	$6.7	$7.4	$7.6	$7.7	$7.9	$8.1	$8.3	$8.6	$9.0	$9.3	$9.7	$10.1	$10.5	$10.9	$11.3	$11.7	$12.1
Residential	$2.9	$2.9	$3.0	$3.0	$3.1	$3.1	$3.2	$3.3	$3.5	$3.6	$3.7	$3.9	$4.0	$4.1	$4.3	$4.5	$4.6
Other	$3.7	$4.5	$4.6	$4.7	$4.8	$4.9	$5.1	$5.3	$5.5	$5.7	$6.0	$6.2	$6.5	$6.7	$7.0	$7.3	$7.5
Energy Taxes ($ Millions)	$1,078.8	$932.6	$897.6	$856.6	$869.5	$884.8	$904.6	$927.3	$952.9	$980.8	$1,010.6	$1,041.8	$1,073.4	$1,105.0	$1,136.7	$1,169.0	$1,198.5
Total - TEFA	$858.8	$822.6	$842.6	$856.6	$869.5	$884.8	$904.6	$927.3	$952.9	$980.8	$1,010.6	$1,041.8	$1,073.4	$1,105.0	$1,136.7	$1,169.0	$1,198.5
Sales	$697.1	$630.1	$639.4	$647.8	$658.5	$670.8	$686.5	$704.6	$724.5	$745.8	$768.5	$792.4	$816.6	$841.3	$866.7	$892.9	$916.1
Corporate Business	$161.7	$192.5	$203.2	$208.8	$211.0	$214.0	$218.1	$222.7	$228.4	$235.0	$242.1	$249.4	$256.8	$263.6	$270.0	$276.1	$282.4
Transitional Facility Assessment	$220.0	$110.0	$55.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Gross State Product for Utilities ($ Billions)	$8.04	$8.16	$8.39	$8.52	$8.56	$8.63	$8.72	$8.82	$8.94	$9.09	$9.24	$9.40	$9.56	$9.71	$9.85	$9.98	$10.12
Gross State Product ($Billions 2000=100)	$354.56	$364.07	$373.80	$383.94	$393.60	$403.28	$414.13	$426.02	$438.82	$452.81	$467.91	$483.79	$500.38	$517.73	$535.83	$554.89	$575.05
Employment at Utilities (Thousands)	15.4	15.3	15.4	15.4	15.4	15.3	15.3	15.3	15.3	15.3	15.2	15.2	15.2	15.2	15.2	15.2	15.2
Electric Utitlies	8.9	8.8	8.7	8.6	8.6	8.5	8.5	8.4	8.4	8.4	8.4	8.3	8.3	8.3	8.3	8.3	8.3
Other	6.4	6.6	6.7	6.8	6.8	6.8	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9
NonAgricultural Employment (Thousands)	4030.1	4079.6	4133.7	4183.5	4227.0	4270.2	4317.3	4363.0	4411.5	4462.8	4521.2	4576.0	4627.0	4676.6	4729.0	4783.0	4843.0
NJ Consumer Price Index (1982-84=100) [1]	196.8	199.4	201.8	205.3	209.5	214.0	218.9	224.5	230.2	235.9	241.7	247.8	254.0	260.3	266.8	273.5	280.2
Renewable Portfolio Standard	3.25	3.40	4.13	5.12	6.21	7.22	8.44	9.67	10.90	12.13	13.35	14.58	15.81	17.03	18.26	19.49	20.00
Class 1: Photovoltaics, et al.	0.75	0.90	1.63	2.62	3.71	4.72	5.94	7.17	8.40	9.63	10.85	12.08	13.31	14.53	15.76	16.99	17.50
Class 2:Hydroelectric and Waste-to-Energy	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50

1 The NJ CPI is a population-weighted average of the CPIs for NY-NJ-CT and PA-NJ.

Table A.8: R/ECON BPU BASELINE WITH HIGH PPI ENERGY

Electric Utilities: Prices, Usage, Taxes, etc.	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	Growth 2004 - 2020
Electric Price per Kilowatt Hour	$0.100	$0.101	$0.101	$0.102	$0.103	$0.104	$0.105	$0.106	$0.107	$0.107	$0.108	$0.109	$0.110	$0.111	$0.112	$0.112	$0.113	13.0%
Residential	$0.111	$0.111	$0.111	$0.111	$0.112	$0.113	$0.114	$0.115	$0.116	$0.116	$0.117	$0.118	$0.119	$0.120	$0.121	$0.122	$0.123	10.8%
Other	$0.093	$0.095	$0.096	$0.097	$0.099	$0.100	$0.100	$0.101	$0.102	$0.103	$0.103	$0.104	$0.105	$0.106	$0.107	$0.108	$0.109	17.2%
Electricity Usage in 1000 Megawatt Hours	66,537	73,628	75,441	76,576	77,582	78,600	79,613	80,608	81,625	82,671	83,786	84,943	86,055	87,141	88,233	89,358	90,529	36.1%
Residential	26,367	26,537	26,940	27,398	27,856	28,309	28,753	29,192	29,630	30,071	30,521	30,981	31,449	31,921	32,396	32,871	33,348	26.5%
Other	40,170	47,091	48,501	49,178	49,726	50,291	50,860	51,416	51,995	52,600	53,265	53,963	54,606	55,220	55,837	56,487	57,181	42.3%
Electric Revenues ($ Billions)	$6.7	$7.4	$7.6	$7.8	$8.0	$8.2	$8.4	$8.6	$8.7	$8.9	$9.1	$9.3	$9.5	$9.7	$9.9	$10.1	$10.3	55.1%
Residential	$2.9	$2.9	$3.0	$3.0	$3.1	$3.2	$3.3	$3.4	$3.4	$3.5	$3.6	$3.7	$3.7	$3.8	$3.9	$4.0	$4.1	40.2%
Other	$3.7	$4.5	$4.7	$4.8	$4.9	$5.0	$5.1	$5.2	$5.3	$5.4	$5.5	$5.6	$5.7	$5.9	$6.0	$6.1	$6.2	66.8%
Energy Taxes ($ Millions)	$1,108.3	$933.0	$899.7	$860.7	$876.6	$891.7	$906.7	$922.1	$938.5	$955.9	$974.2	$993.3	$1,012.3	$1,030.9	$1,049.3	$1,068.0	$1,087.4	-1.9%
Energy Taxes - TEFA	$858.8	$823.0	$844.7	$860.7	$876.6	$891.7	$906.7	$922.1	$938.5	$955.9	$974.2	$993.3	$1,012.3	$1,030.9	$1,049.3	$1,068.0	$1,087.4	26.6%
Sales	$697.1	$630.6	$642.6	$653.9	$667.5	$679.0	$689.3	$699.5	$709.9	$720.5	$731.8	$743.6	$755.3	$767.1	$779.2	$791.8	$805.0	15.5%
Corporate Business	$161.7	$192.4	$202.1	$206.8	$209.1	$212.7	$217.4	$222.6	$228.6	$235.3	$242.4	$249.7	$257.0	$263.7	$270.1	$276.2	$282.5	74.6%
Transitional Facility Assessment	$249.5	$110.0	$55.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	
Gross State Product for Utilities ($ Billions)	$8.04	$8.16	$8.37	$8.47	$8.52	$8.60	$8.70	$8.82	$8.95	$9.09	$9.25	$9.41	$9.56	$9.71	$9.85	$9.98	$10.12	25.9%
Gross State Product ($Billions 2000=100)	$354.57	$364.09	$373.80	$383.92	$393.57	$403.25	$414.14	$426.08	$438.96	$453.02	$468.20	$484.16	$500.85	$518.29	$536.47	$555.62	$575.88	62.4%
Employment at Utilities (Thousands)	15.4	15.3	15.4	15.4	15.4	15.3	15.3	15.3	15.3	15.3	15.2	15.2	15.2	15.2	15.2	15.2	15.2	-1.2%
Electric Utilities	8.9	8.8	8.7	8.6	8.6	8.5	8.5	8.4	8.4	8.4	8.4	8.3	8.3	8.3	8.3	8.3	8.3	-6.4%
Other	6.4	6.6	6.7	6.8	6.8	6.8	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	7.7%
NonAgricultural Employment (Thousands)	4030.2	4079.7	4133.8	4183.6	4227.1	4270.2	4317.4	4363.2	4411.9	4463.4	4521.9	4576.9	4628.1	4677.9	4730.5	4784.8	4845.0	20.2%
NJ Consumer Price Index (1982-84=100)[1]	196.6	199.4	201.9	205.4	209.5	213.9	218.7	224.2	229.8	235.3	241.1	247.1	253.2	259.4	265.8	272.4	279.1	41.9%
Renewable Portfolio Standard	3.25	3.40	4.13	5.12	6.21	6.50	6.50	6.50	6.50	6.50	6.50	6.50	6.50	6.50	6.50	6.50	6.50	100.0%
Class 1: Photovoltaics, et al.	0.75	0.90	1.63	2.62	3.71	4.00	4.00	4.00	4.00	4.00	4.00	4.00	4.00	4.00	4.00	4.00	4.00	433.3%
Class 2: Hydroelectric and Waste-to-Energy	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	0.0%

1 The NJ CPI is a population-weighted average of the CPIs for NY-NJ-CT and PA-NJ.

Table A.9: R/ECON BPU STRAIGHTLINE RPS INCREASE 2009 to 20% IN 2020
OUT-OF-STATE MANUFACTURING OF RENEWABLE TECHNOLOGIES

Electric Utilities: Prices, Usage, Taxes, etc.	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020
Electric Price per Kilowatt Hour	$0.100	$0.100	$0.100	$0.100	$0.101	$0.102	$0.102	$0.104	$0.104	$0.106	$0.107	$0.108	$0.108	$0.109	$0.110	$0.111	$0.112
Residential	$0.111	$0.111	$0.110	$0.110	$0.110	$0.111	$0.111	$0.112	$0.113	$0.114	$0.114	$0.115	$0.116	$0.117	$0.118	$0.119	$0.120
Other	$0.093	$0.095	$0.095	$0.095	$0.097	$0.097	$0.098	$0.099	$0.100	$0.102	$0.103	$0.103	$0.104	$0.104	$0.106	$0.107	$0.108
Electricity Usage in 1000 Megawatt Hours	66,537	73,645	75,484	76,618	77,625	78,611	79,622	80,566	81,617	82,592	83,781	84,939	86,059	87,162	88,214	89,358	90,535
Residential	26,367	26,538	26,946	27,405	27,864	28,308	28,753	29,193	29,625	30,068	30,520	30,974	31,444	31,920	32,390	32,867	33,347
Other	40,170	47,107	48,538	49,213	49,761	50,302	50,869	51,372	51,993	52,524	53,261	53,964	54,615	55,242	55,825	56,490	57,188
Electric Revenues ($ Billions)	$6.7	$7.4	$7.6	$7.7	$7.9	$8.0	$8.2	$8.4	$8.5	$8.8	$9.0	$9.1	$9.3	$9.5	$9.7	$10.0	$10.2
Residential	$2.9	$2.9	$3.0	$3.0	$3.1	$3.1	$3.2	$3.3	$3.3	$3.4	$3.5	$3.6	$3.6	$3.7	$3.8	$3.9	$4.0
Other	$3.7	$4.5	$4.6	$4.7	$4.8	$4.9	$5.0	$5.1	$5.2	$5.4	$5.5	$5.6	$5.7	$5.7	$5.9	$6.0	$6.2
Energy Taxes ($ Millions)	$1,108.3	$932.6	$897.7	$856.7	$869.5	$881.9	$894.9	$910.9	$927.2	$949.0	$966.8	$986.2	$1,004.5	$1,021.0	$1,041.0	$1,059.8	$1,081.3
Total - TEFA	$858.8	$822.6	$842.7	$856.7	$869.5	$881.9	$894.9	$910.9	$927.2	$949.0	$966.8	$986.2	$1,004.5	$1,021.0	$1,041.0	$1,059.8	$1,081.3
Sales	$697.1	$630.1	$639.4	$647.8	$658.5	$667.9	$676.8	$688.1	$698.7	$713.8	$724.4	$736.4	$747.4	$757.0	$770.7	$783.3	$798.6
Corporate Business	$161.7	$192.6	$203.3	$208.9	$211.0	$214.0	$218.1	$222.9	$228.5	$235.3	$242.4	$249.8	$257.1	$264.0	$270.3	$276.5	$282.7
Transitional Facility Assessment	$249.5	$110.0	$55.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Gross State Product for Utilities ($ Billions 2000=100)	$8.13	$8.26	$8.50	$8.62	$8.67	$8.73	$8.82	$8.93	$9.05	$9.20	$9.36	$9.52	$9.68	$9.83	$9.97	$10.11	$10.25
Gross State Product ($Billions 2000=100)	$380.81	$391.04	$401.50	$412.39	$422.76	$433.15	$444.83	$457.63	$471.43	$486.52	$502.81	$519.94	$537.86	$556.60	$576.13	$596.71	$618.47
Employment at Utilities (Thousands)	15.4	15.3	15.4	15.4	15.4	15.3	15.3	15.3	15.3	15.3	15.2	15.2	15.2	15.2	15.2	15.2	15.2
Electric Utitlies	8.9	8.8	8.7	8.6	8.6	8.5	8.5	8.4	8.4	8.4	8.4	8.3	8.3	8.3	8.3	8.3	8.3
Other	6.4	6.6	6.7	6.8	6.8	6.8	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9
NonAgricultural Employment (Thousands)	4030.2	4079.8	4134.0	4183.9	4227.6	4271.4	4317.9	4363.5	4412.5	4463.9	4522.5	4577.5	4629.1	4678.9	4731.7	4786.3	4846.8
NJ Consumer Price Index (1982-84=100)[1]	196.6	199.4	201.8	205.3	209.5	213.9	218.8	224.3	229.9	235.4	241.2	247.2	253.3	259.5	265.9	272.5	279.2
Renewable Portfolio Standard	3.25	3.40	4.13	5.12	6.21	7.22	8.44	9.67	10.90	12.13	13.35	14.58	15.81	17.03	18.26	19.49	20.00
Class 1: Photovoltaics, et al.	0.75	0.90	1.63	2.62	3.71	4.72	5.94	7.17	8.40	9.63	10.85	12.08	13.31	14.53	15.76	16.99	17.50
Class 2:Hydroelectric and Waste-to-Energy	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50

1 The NJ CPI is a population-weighted average of the CPIs for NY-NJ-CT and PA-NJ.

Table A.10: R/ECON BPU STRAIGHTLINE RPS INCREASE 2009 to 20% IN 2020 WITH MANUFACTURING AND MAINTENANCE OF NEW TECHNOLOGIES INSTATE

R/ECON BPU STRAIGHTLINE RPS INCREASE 2009 to 20% IN 2020 WITH MANUFACTURING AND MAINTENANCE OF NEW TECHNOLOGIES INSTATE																	
Electric Utilities: Prices, Usage, Taxes, etc.	**2004**	**2005**	**2006**	**2007**	**2008**	**2009**	**2010**	**2011**	**2012**	**2013**	**2014**	**2015**	**2016**	**2017**	**2018**	**2019**	**2020**
Electric Price per Kilowatt Hour	$0.100	$0.100	$0.100	$0.100	$0.101	$0.102	$0.102	$0.104	$0.104	$0.106	$0.107	$0.108	$0.108	$0.109	$0.110	$0.111	$0.112
Residential	$0.111	$0.111	$0.110	$0.110	$0.110	$0.111	$0.111	$0.112	$0.113	$0.114	$0.114	$0.115	$0.116	$0.117	$0.118	$0.119	$0.120
Other	$0.093	$0.095	$0.095	$0.095	$0.097	$0.097	$0.098	$0.099	$0.100	$0.102	$0.103	$0.103	$0.104	$0.104	$0.106	$0.107	$0.108
Electricity Usage in 1000 Megawatt Hours	66,537	73,648	75,494	76,629	77,651	78,664	79,679	80,611	81,674	82,658	83,852	85,019	86,153	87,263	88,325	89,486	90,679
Residential	26,367	26,538	26,946	27,406	27,865	28,310	28,756	29,198	29,630	30,073	30,527	30,981	31,452	31,929	32,399	32,878	33,359
Other	40,170	47,110	48,548	49,223	49,786	50,354	50,923	51,413	52,045	52,585	53,326	54,038	54,701	55,335	55,926	56,608	57,320
Electric Revenues ($ Billions)	$6.7	$7.4	$7.6	$7.7	$7.9	$8.0	$8.2	$8.4	$8.6	$8.8	$9.0	$9.1	$9.3	$9.5	$9.8	$10.0	$10.2
Residential	$2.9	$2.9	$3.0	$3.0	$3.1	$3.1	$3.2	$3.3	$3.3	$3.4	$3.5	$3.6	$3.6	$3.7	$3.8	$3.9	$4.0
Other	$3.7	$4.5	$4.6	$4.7	$4.8	$4.9	$5.0	$5.1	$5.2	$5.4	$5.5	$5.6	$5.7	$5.8	$5.9	$6.1	$6.2
Energy Taxes ($ Millions)	$1,108.3	$932.7	$897.8	$856.8	$869.7	$882.3	$895.4	$911.4	$927.7	$949.6	$967.3	$986.8	$1,005.2	$1,021.7	$1,041.9	$1,060.7	$1,082.4
Total - TEFA	$858.8	$822.7	$842.8	$856.8	$869.7	$882.3	$895.4	$911.4	$927.7	$949.6	$967.3	$986.8	$1,005.2	$1,021.7	$1,041.9	$1,060.7	$1,082.4
Sales	$697.1	$630.1	$639.5	$647.9	$658.7	$668.3	$677.2	$688.4	$699.0	$714.2	$724.9	$737.0	$748.0	$757.7	$771.4	$784.2	$799.5
Corporate Business	$161.7	$192.6	$203.3	$208.9	$211.0	$214.1	$218.2	$223.0	$228.7	$235.4	$242.5	$249.8	$257.2	$264.1	$270.5	$276.6	$282.9
Transitional Facility Assessment	$249.5	$110.0	$55.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Gross State Product for Utilities ($ Billions 2000=100)	$8.13	$8.26	$8.50	$8.62	$8.67	$8.73	$8.83	$8.93	$9.06	$9.20	$9.36	$9.52	$9.69	$9.84	$9.98	$10.11	$10.25
Gross State Product ($Billions 2000=100)	$380.81	$391.06	$401.57	$412.50	$422.93	$433.40	$445.16	$458.02	$471.89	$487.03	$503.37	$520.57	$538.55	$557.34	$576.95	$597.61	$619.4542
Employment at Utilities (Thousands)	15.4	15.3	15.4	15.4	15.4	15.4	15.3	15.3	15.3	15.3	15.3	15.2	15.2	15.2	15.2	15.2	15.2
Electric Utitlies	8.9	8.8	8.7	8.6	8.6	8.5	8.5	8.4	8.4	8.4	8.4	8.3	8.3	8.3	8.3	8.3	8.3
Other	6.4	6.6	6.7	6.8	6.8	6.8	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9	6.9
NonAgricultural Employment (Thousands)	4030.2	4080.5	4134.8	4185.1	4231.0	4276.4	4321.4	4367.2	4417.3	4469.1	4528.1	4584.2	4636.6	4686.7	4740.7	4796.7	4858.2
NJ Consumer Price Index (1982-84=100)[1]	196.6	199.4	201.8	205.3	209.5	213.9	218.8	224.3	229.9	235.4	241.2	247.2	253.3	259.5	265.9	272.5	279.2
Renewable Portfolio Standard	3.25	3.40	4.13	5.12	6.21	7.22	8.44	9.67	10.90	12.13	13.35	14.58	15.81	17.03	18.26	19.49	20.00
Class 1: Photovoltaics, et al.	0.75	0.90	1.63	2.62	3.71	4.72	5.94	7.17	8.40	9.63	10.85	12.08	13.31	14.53	15.76	16.99	17.50
Class 2:Hydroelectric and Waste-to-Energy	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50	2.50

1 The NJ CPI is a population-weighted average of the CPIs for NY-NJ-CT and PA-NJ.

Appendix B:
Supplement to the Environmental Externality Analysis

SUPPLEMENT TO THE ENVIRONMENTAL EXTERNALITY ANALYSIS

1. INTRODUCTION

The purpose of this appendix is to expand upon the environmental externality discussion in Chapter 3.

This appendix uses a benefit transfer approach. This method consists of conducting a thorough literature review of recent, North American applicable, peer-reviewed studies. The key question in following this approach is how closely other studies resemble the situation in New Jersey based on timeliness, methodology, and other factors. Ideally, primary research based on the characteristics of New Jersey is preferred to a benefit transfer approach, but the types of benefits are too numerous and their phenomenon too complex to pursue primary research given the financial and time constraints of this project.

The general steps taken to estimate non-market benefits of air pollution abatement are as follows:

1. Identifying Benefits
2. Quantifying Benefits
3. Monetizing Benefits

The first step involves describing a qualitative relationship between changes in pollutant emissions and ambient concentrations, and subsequently between ambient concentrations and environmental effects.

Identifying the benefits of air pollution abatement is equivalent to identifying the damages that are reduced or avoided. These damages fall into three broad categories (adopted from Freeman, 1993):

1. Direct damages to humans
2. Indirect damages to humans through ecosystems
3. Indirect damages to humans through nonliving systems

Direct damages to humans include health damages, as well as aesthetic damages such as unpleasant odor, noise or poor visibility. Indirect damages to humans through ecosystems consist of productivity damages in the form of crop reduction, and damages to forests and commercial fisheries; recreation damages (lakes, rivers, etc.), and intrinsic or nonuse damages. The latter are damages to ecological resources that are not motivated by people's own use of these resources. For example, people value endangered species or rare ecosystems, even though they do not have the intention to ever see or experience them. Finally, indirect damages to humans that occur through nonliving things include damages to materials and structures, such as soiling and corrosion.

The second step, quantifying benefits, involves establishing a functional relationship between environmental effects and the reduction in air pollution. For example, such

quantitative relationships can be described by dose-response or concentration-response functions. These functions describe the change in a health effect, say, asthma attacks, and the concentration of the pollutant that causes the effect. In order to calculate the number of cases that will be avoided, we need to establish a baseline exposure (number of people affected, and the level of pollution they are subjected to) and the baseline number of cases for each quantifiable health effect for each pollutant. These numbers are then contrasted with the number of cases for each quantifiable health effect with the regulation (RPS, in our case), to calculate the number of cases avoided as a result of the RPS.

The third step, monetizing or valuing the benefits, is typically specific to the environmental effect under consideration. In what follows, we describe separately the most common valuation methods used in the literature for each effect.

As part of the process of evaluating the evidence presented by this body of literature, each study must be evaluated as to the soundness of the data, the analytic techniques and the conclusions drawn by the authors.

Although the approach of this report is to determine whether these non-market benefits can be monetized in the context of NJ RPS policymaking, there are other ways of accounting for these benefits in policymaking without monetization. One method is tradeoff analysis, and another involves a deliberation process. If tradeoff analysis were to be applied here, then each of the non-market benefits would be quantified separately and not combined into a dollar value. Tradeoffs between different impacts, such as costs and illnesses due to sulfur dioxide emissions, would be evaluated. Such an analysis can also be informed by stakeholders' values pertaining to the relative importance of different impacts have. If we used a deliberative process, for example one mandated by law, various stakeholders would provide input. An outcome is considered successful if the process is successfully completed.

2. CRITERIA FOR BENEFIT TRANSFER

Instead of conducting primary research, valuation studies often apply the benefit transfer method. Benefit transfer entails using monetary values estimated in existing empirical studies to assess the value of a quantified effect in a different study.

When is Benefit Transfer applicable?

1. When existing studies value similar effects.
2. When the context of the existing studies is highly similar.
3. When the existing studies are of high quality.

There are no universally accepted criteria for benefit transfer, but in most cases the defensibility of the benefit estimates depends on the quality of the existing study. Hence one criterion for benefit transfer is to select studies that were published in peer-reviewed journals, and have been viewed highly by the professional community.

Benefit transfer may increase the inherent uncertainty surrounding environmental benefits estimates, but for practical reasons (e.g. cost and time), benefit transfer is a necessary component of policy analysis. In most situations, the question is not whether to conduct benefit transfer, but how to improve benefit transfer to make it more reliable.

When one uses benefit transfer, there are three sources of variation between the original study location/situation and the one to which the benefits estimates are transferred to that one has to consider. The first is individual variation (x), the second is commodity variation (y), and the third is other variation (z).

Consider the following hypothetical situation. Suppose that the state of New Jersey is planning to upgrade a state park, and one proposal is to provide a swimming beach to the lake. The benefit subject to valuation is recreational swimming at the lake. Suppose researchers have identified studies from other parts of the country that value similar benefits. The estimates of recreational swimming benefits may not accurately measure the benefit of recreational swimming in New Jersey for three main reasons: 1. The preferences of New Jersey residents may differ from the preferences of the participants of the selected studies. This is the individual variation (x) between the participants of the original study, and the population to which benefit transfer is applied. 2. The characteristics of the benefit may also differ. For example, the benefit derived from the availability of a clean beach for swimming may be different in Alaska as it is in New Jersey. This is called the commodity variation (y). 3. The first two sources of variation imply that the value of recreational swimming in New Jersey is different from the context of the original study, while the third source of variation (z) implies that the value was incorrectly estimated. Each type of variation has a fixed and a random component (see table below).

	Individual Variation x	Commodity Variation y	Other Variation z
Fixed Component	μ_I	μ_C	μ_O
Random Component	ε_I	ε_C	ε_O

The fixed component of each variation includes variables that can be measured and observed by the researchers, while random variations are not observable. Hence, we can make benefit transfer more reliable by minimizing the fixed component of each variation. Individual variation may be reduced, if one is able to estimate how the value of recreational swimming varies with demographic and socioeconomic variables, such as age, income, etc. Using the data on these observable variables, one may adjust the estimates for the policy context. Similarly, if one identifies the relationship between the benefits provided by the different commodities, then the second type of variation is reduced. For example, one might conduct a small calibration survey for the policy site. The results from the original study could then be weighted by the respondents' attitude/experience values for the policy site to calibrate the transfer. Selecting high-quality studies for the policy context minimizes the third type of variation. The main criteria in study selection should include statistical tests of the explanatory power and robustness of the models used in estimation. The calibration of the original estimates may reduce the variation between the policy context and the original study, and hence enhance the defensibility of benefit transfer.

3. DIRECT BENEFITS TO HUMANS – HEALTH BENEFITS

Linkages between air pollution and health effects are subtle and often difficult to establish. However, the available literature provides strong evidence that air pollution has adverse effects on human health. In general, the information that is needed for the estimation of health benefits is similar to the input needed for estimating non-health related benefits of air pollution abatement. The first step is to estimate the change in emissions resulting from the regulatory action. The next step involves air quality modeling to estimate the relationship between changes in emissions and changes in ambient pollutant concentrations. Third, one must determine the population and risk, in order to be able to estimate the health effects. Fourth, the health effects must be monetized to conduct a cost-benefit analysis.

3.1 IDENTIFYING HEALTH BENEFITS

Health benefits resulting from reduced air pollution can be grouped into two broad categories: mortality benefits and morbidity benefits. Mortality benefits are avoided deaths from diseases caused by air pollution. Morbidity benefits refer to avoided cases of non-fatal health effects. Air pollutants that have been linked to adverse health effects include particulate matter (PM), ozone (O_3), carbon monoxide (CO), sulfur dioxide (SO_2), and nitrogen dioxide (NO_2). Until the mid-1980's it was believed that ambient pollutant concentrations did not have adverse health effects (Katsouyanni, 2003). Over the last 15 years, a large body of epidemiological literature has been devoted to the study of adverse health effects occurring at moderate and low pollutant concentrations. There is now sufficient evidence to support the hypothesis that both chronic and acute health effects can occur at ambient pollution levels. Current research focuses on the consequence of acute and chronic air pollution exposure for excess cardiovascular and respiratory morbidity and mortality.

Types of health studies

There are two main types of methods to establish a dose-response relationship for a health effect:

1. Epidemiological studies (cohort studies, case control studies, occupational epidemiology studies, cross-sectional studies)
2. Toxicological studies (mechanistic studies, animal studies, human studies)

Epidemiological studies attempt to establish a quantitative relationship between health effect and air pollution using a sample drawn from a large population. The four most commonly used types of epidemiological studies are cohort studies, case control studies, occupational epidemiology studies, and cross-sectional studies.

Cohort studies follow a group of healthy people with different levels of exposure and assess the impact of exposure on their health over time. The term cohort in epidemiology refers to a collection of people that share some common characteristics, such as age or ethnicity. The two common types of cohort studies, prospective and retrospective, both start by identifying and enrolling subjects based upon the presence or absence of exposure, without knowing whether the exposure resulted in any adverse impact. In a typical prospective cohort study, individuals in a cohort are followed forward in time, for a sufficiently long period, to track the appearance of a disease and disability. On the contrary, in retrospective cohort studies first the cohorts are selected and then the exposure histories of the participants are collected and studied. Cohort studies are used because in this setting the issue of temporality is controlled, since the exposure precedes the disease process. Besides producing more reliable estimates, prospective cohort studies also allow us to study how multiple risk factors determine the onset and history of one or more diseases.

Case control studies identify a group of individuals with a certain health condition, as well as a group of subjects without the health effect (control group), and try to answer the question why some people got ill, while the those in the control group did not. Case control studies are less time-intensive and expensive than cohort studies, but they are also subject to a greater estimation bias.

Occupational epidemiology studies people working in particular jobs as subjects. Workers in certain occupations often have a higher exposure to a pollutant than the general population, and hence it may be easier to identify a causal relationship between exposure to a pollutant and the health effect. Occupational epidemiology studies may not be appropriate for benefit transfer because their study populations may not represent the general populations in terms of risk.

Cross-sectional studies analyze the relationship between a group's (e.g. a metropolitan area) health status and exposure status simultaneously. This study design does not allow for changes in variables over time, and hence it may fail to uncover the true relationship between exposure and a health outcome.

In toxicology, mechanistic studies examine how and why various disease processes occur in response to toxicant exposures, and help establish a relationship between dose or exposure and response. Animal studies also provide precise information about the adverse response to a substance because the studies are controlled in a laboratory, and animals are subjected to a wide range of exposures. One of the advantages of animal studies is the researcher's ability to extrapolate from the high doses in animal studies down to the low doses often experienced in human exposure scenarios. Finally, human studies can be used to extrapolate the response of humans at low doses to higher doses.

Long-term (chronic) health effects of air pollution have been evaluated by a large number of cross-sectional and a few prospective-cohort studies (Dockery et al., 1993; Pope et al., 1995; HEI, 2000; Pope et al., 2002). Prospective cohort study is the desirable design because exposure precedes the health outcome, which is a necessary condition for

establishing a causal relationship between exposure and the health outcome. Moreover, this study design is less subject to bias because exposure is evaluated before the health status is known, and also more accurate data may be collected. Künzli and Tager (2000) argue that cross-sectional and prospective cohort studies address different aspects of the association between air pollution and mortality. Cross-sectional studies are only capable of capturing mortality effects triggered by air pollution exposures that occurred shortly before death, while prospective cohort studies capture all air pollution-related mortality effects.

Health benefits due to particulate matter reductions

Adverse health effects of exposure to particles have been described in numerous epidemiological studies. Health endpoints include all-cause and cause-specific mortality and hospital admissions. Studies conducted in the United States and in other countries have reported associations between changes in PM and changes in mortality and morbidity, particularly among subgroups of people with respiratory or cardiovascular diseases. However, the exact mechanisms by which PM influences human health are not well understood. Earlier literature focused on PM greater than 10μm in diameter, while in the last decade the attention of researchers turned to fine particles such as $PM_{2.5}$. Recent research indicates that ultrafine particles (UF) less than 0.1μm in diameter may play an important role in the induction of toxic effects. Currently, however, data on UF exposure and health effects are still limited.

Although the importance of long-term exposure to PM has been emphasized, most of the attention in the literature has been devoted to short-term health effects. Two prominent prospective-cohort studies of mortality effects of PM are Dockery et al. (1993) and Pope et al. (1995). Both of these are prospective cohort studies. Unlike earlier studies, Dockery et al. (1993) estimate the effect of air pollution on mortality while controlling for individual risk factors. Pope et al. (1995) study the association between air pollution and mortality using data from a large cohort drawn from many study areas. Pope et al. (2002) is a continuation of the Pope et al. (1995), while HEI (2000) study is a reanalysis of the original Pope et al. (1995) data. Information on data, methodology, and the results of these four studies are summarized in Table 3.1.

Short-term, or acute effects of PM are well established for morbidity endpoints such as, hospital admissions for respiratory and cardiovascular conditions. There is also evidence of acute effects on respiratory function, lower respiratory symptoms, and increased medication use by asthmatics (Katsouyanni, 2003). There are fewer studies available on the long-term, or chronic, health effects of PM pollution. A few studies have linked an increase in chronic bronchitis occurrence to an increase in ambient PM concentration (Abbey et al., 1993; Schwartz, 1993; Abbey et al., 1995). Tables 3.6-3.9, at the end of this chapter, summarize the available studies that assessed morbidity effects resulting in chronic and minor illness, as well as hospital admissions. We selected studies that were conducted in the United States and Canada, and found a positive association between the health effect and air pollution.

Table 3.1. Prospective Cohort Studies of Mortality Due to Particulate Matter Pollution			
Study	Study Location and Population	Study Period	Results
Dockery et al. (1993)	8111 adults in six U.S. cities	14-to-16-year mortality follow-up	Air pollution was positively associated with death from lung cancer and cardiopulmonary disease but not with death from other causes considered together. Mortality was most strongly associated with air pollution with fine particulates, including sulfates.
Pope et al. (1995)	552,138 adults in 151 U.S. metropolitan areas	Ambient pollution data: 1980 Enrollment: 1982 Deaths through: 1989	PM pollution associated with cardiopulmonary and lung cancer mortality but not with mortality due to other causes. Increased mortality is associated with sulfate and fine particulate air pollution at levels commonly found in U.S. cities.
HEI (2000)	552,138 adults in 151 U.S. metropolitan areas	Ambient pollution data: 1980 Enrollment: 1982 Deaths through: 1989	The original results of Dockery et al. (1993) and Pope et al. (1995) were successfully replicated and validated. In alternative models, estimated mortality effects increased in the subgroup of subjects with less than high school education. When sulfur dioxide was included in models with fine particles or sulfate, the associations between these pollutants (fine particles and sulfate) and mortality diminished.
Pope et al. (2002)	319,000-590,000 adults in 51-102 U.S. metropolitan areas, depending on the PM measure and study period	Enrollment: 1982 Deaths through: 1998	Fine particle and sulfur oxide pollution were associated with all-cause death, lung cancer and cardiopulmonary mortality. Each 10$\mu g/m^3$ increase in fine PM pollution was associated with approximately 4%, 6%, and 8% increase in the risk of all-cause death, cardiopulmonary mortality, and lung cancer mortality, respectively.

Health benefits due to ozone reductions

Ozone is formed by a chemical reaction from its precursor pollutants (volatile organic compounds (VOCs) and nitrogen oxides (NO, NO_2), and nitrous oxide (N_2O)) in the presence of heat and sunlight. Ozone concentration is the highest in the summer when the weather is hot and sunny with relatively light winds. Electric power plants are among the main sources of precursors pollutant emissions.

Health problems are caused by tropospheric, or ground-level, ozone. Ozone is associated with a variety of adverse health effects ranging from minor symptoms to hospital admissions and chronic illness. Some studies have found a link between ozone and mortality, however there is significant uncertainty about the relationship between mortality and high ozone concentrations, partly because of the possible confounding effect of other pollutants such as particulate matter. Table 3.2 below summarizes the most common adverse health effects associated with ozone.

Table 3.2. Likely Ozone-related Adverse Health Effects

Adverse Health Effect	Comment
Respiratory Hospital Admissions	A large number of studies have linked ozone to hospital admissions for pneumonia, chronic obstructive pulmonary disease (COPD), asthma and other respiratory ailments.
Cardiovascular Hospital Admissions	There is a link between high ozone and dysrhythmias (abnormal heartbeat patterns).
Total Respiratory ER Visits	Studies have also found a link between high ozone and emergency room visits which do not result in actual hospital admissions.
Minor Symptoms	Short-term exposure to ozone has been linked to a variety of symptoms, including cough, sore throat and head cold.
Asthma Attacks	Ozone has specifically been linked to incidence of asthma attacks and may be linked to the development of chronic asthma.
Shortness of breath	Ozone associated with shortness of breath in asthmatics and non-asthmatics.

Source: Abt Associates (1999)

Mechanistic studies of ozone yield a sufficient evidence for a biologic plausibility of respiratory-related morbidity and mortality. For a review of mechanistic studies of ozone see Levy et al. (2001). There is evidence from human and animal exposure studied that long-term exposure to ozone may cause a sustained decrement in lung function. There are well-documented molecular mechanisms for acute respiratory effects of ozone, but the

evidence for chronic respiratory effects is limited. There is, however, increasing evidence that high ozone can result in the development of chronic diseases. For example, McConnell (2002) provided the first evidence suggesting that tropospheric ozone causes the development of childhood asthma. In high ozone-concentration cities, children who played outdoor sports were 3 to 4 times more likely to develop chronic asthma than children who did not play sports. In low ozone-concentration cities, children playing sports were no more likely to develop asthma than children who did not play sports.

Because indoor ozone concentrations are generally lower than ambient concentrations, personal exposure may not be directly related to ambient concentration. Personal exposures to ozone are influenced by air conditioning or averting behavior, such as, more time spent indoors. When applying concentration-response functions, one must determine the relationship between ambient ozone concentrations and personal exposures. An understanding of any systemic differences between the study and policy region is crucial.

The importance of personal exposures to ozone led to new recent stream of research where the analysis of health effects is stratified by some relevant personal characteristics (e.g. insurance status) or regional characteristics (e.g. prevalence of air conditioning). Two recent studies, Gwynn and Thurston (2001) and Nauenberg and Basu (1999) find that insurance status as a factor in the strength of the association between ozone and hospital admissions for asthma. Jaffe et al. (2003) adjust for insurance status in the relationship between air pollution and emergency department (ED) visits, by looking at asthma ED visits and ozone among Medcaid recipients. Finkelstein et al. (2000) find that among predictors of emergency room visits for asthma is insurance status.

Health benefits due to carbon monoxide reductions

Carbon monoxide (CO) is a colorless and odorless gas produced through incomplete combustion of carbon-based fuels. Carbon monoxide enters the bloodstream through the lungs and reduces the delivery of oxygen to the body's organs and tissues. The most vulnerable to CO are those who suffer from cardiovascular disease, particularly those with angina or peripheral vascular disease. Fetuses and young infants, children, pregnant women, individuals with obstructive pulmonary disease such as bronchitis and emphysema, smokers, and individuals spending a lot of time on the street working or doing exercise are also more susceptible to CO exposure. In health studies, high CO concentrations have been linked to hospital admissions for asthma, chronic obstructive pulmonary disease (COPD), dsyrhythmias, ischemic heart disease, and congestive heart failure (CHF).

Health benefits due to sulfur dioxide reductions

Sulfur dioxide is formed when fuel, containing sulfur, such as coal and oil, is burned. The main health effects associated with exposure to high SO_2 concentrations include effects on breathing, respiratory illness, changes in pulmonary defenses, and aggravation of cardiovascular disease. The most susceptible groups are children, the elderly, asthmatics,

and people with cardiovascular and chronic lung disease (such as bronchitis and emphysema). High SO_2 levels have been linked to the following endpoints: hospital admissions for pneumonia, ischemic heart disease, and respiratory conditions; chest tightness, shortness of breath, and wheeze.

Health benefits due to nitrogen dioxide reductions

NO_2 is a suffocating, brownish gas that is formed when fuel is burned at high temperatures. Primary sources of NO_2 are motor vehicles, electric utilities and industrial boilers. Nitrogen dioxide can irritate the lungs and lower resistance to respiratory infections such as influenza. There is no clear evidence on the effect of short-term exposure to NO_2 on health, but frequent exposure may cause an increased incidence of acute respiratory illness, especially in children. NO_2 has been linked to hospital admissions for respiratory conditions, pneumonia, congestive heart failure, and ischemic heart disease. Epidemiological studies found that NO_2 has a modifying effect on PM: the increase in mortality due to PM was found to be higher in cities where long-term NO_2 concentrations were higher (Katsouyanni, 2003). In addition NO_2 may have other indirect adverse effects, as it contributes to ozone formation. Therefore the importance of NO_2 for health comes from its role as an O_3 precursor and a contributor to the formation of secondary particles.

3.2 QUANTIFYING HEALTH BENEFITS

Health benefits are typically estimated using the damage-function (DF) method the consists of the following steps involved:

1. Determining the dose-response relationship for each health effect
2. Determining baseline exposure
3. Determining the number of baseline cases for each quantifiable health effect
4. Number exposed × Baseline exposure × Dose-response relationship.
5. Determining exposure after the regulation (for each regulatory option)
6. Determining the number of cases for each quantifiable effect with the regulation
7. Determining the number of cases avoided as a result of each regulatory option

The purpose of quantification is to determine the change in the occurrence of a health effect (y) as a result of a change in pollutant concentrations (x) between the baseline and the control scenario. Such relationship between, say particulate matter (PM) concentration (Δx), and the change in the health effect (Δy) is described by dose-response or concentration-response (CR) functions. Dose-response or concentration-response (CR) functions estimate the risk (of the occurrence of a health effect) per unit of exposure to a pollutant. The two most common functional forms for the CR relationship between air pollutants and a health effect (e.g. mortality) used in the literature are log-linear and linear functions. The linear relationship can be described by the following equation:

$$y = \alpha + \beta \cdot x$$

Where α and β are the parameters to be estimated. From the above equation it follows that:

$$\Delta y = \alpha + \beta \cdot \Delta x$$

Log-linear CR functions have the following general form:

$$y = \gamma \cdot e^{\beta \cdot x}$$

Or, equivalently:

$$\log(y) = \alpha + \beta \cdot x \quad \text{where} \quad \alpha = \log(\gamma).$$

Let y_1 denote the occurrence of the health effect under the baseline scenario, and let y_2 be a measure of the health effect under the control scenario. Then the relationship between the change in PM concentration and the health effect may be written as follows:

$$\Delta y = y_1 - y_2 = -\gamma \cdot e^{\beta \cdot PM_1} \left[\frac{e^{\beta \cdot x_1}}{e^{\beta \cdot x_2}} - 1 \right] = -y_1 \left[e^{-\beta \cdot \Delta x} - 1 \right]$$

In a sufficiently large population, some people develop a disease that can be attributed to air pollution regardless of whether they were exposed or not. Relative risk (RR) is a measure that tells us the seriousness of exposure to a known risk factor. It is defined as the risk is for those exposed relative to those who are not exposed. For example, if the risk of developing a disease in the exposed population is 5%, while in the non-exposed population it is 1%, the relative risk is 5. A high risk factor indicates strong evidence between exposure to a pollutant and the health effect.

The risk factor associated with a log-linear CR function has the following form:

$$RR_{\text{log-linear}} = \frac{y_2}{y_1} = e^{-\beta \cdot \Delta x}$$

Epidemiological studies typically report the relative risk, rather than the CR-coefficients. The above equations may be used to calculate the coefficients from the reported RR-values.

Use of thresholds in CR functions

Typically, dose-response functions that have been estimated for health effects describe a linear no-threshold relationship. This means that every unit of exposure contributes equally to aggregate risk in a large population of people. For example, a linear no-threshold dose-response function treats the case of one person being exposed to one hundred units of the pollutant, and ten people subjected to ten units of pollution equally (simply, as 100 units of human exposure). In some cases, the use of such dose-response functions may not be appropriate. Thresholds may be incorporated into the analysis even when one uses CR-functions that were derived under the no-threshold assumptions. While the possible existence of a threshold in concentration-response relationships is an

important scientific question, there is currently no scientific basis for selecting appropriate threshold levels.

Health studies estimating CR relationship often attempt to justify their linearity assumption. For example, support for the no-threshold assumption in mortality is provided by a recent study by Vedal et al. (2003). They study the association between daily inhalable particle concentrations and daily mortality in Vancouver, British Columbia, where daily average PM_{10} and ozone concentrations have been very low during the study period. After analyzing three years of data, they conclude that increases in low concentrations of air pollution are associated with daily mortality.

To determine the number of baseline cases, we need to identify the segment of population that is exposed, and the number of people exposed within each segment, as well as the level, duration and frequency of exposure. In order to obtain accurate estimates, we also have to control for averting behavior (that is, people with known risk may act to avoid exposure).

Quantifying Mortality Benefits

In valuation studies mortality benefits linked to particulate matter (PM) tend to dominate total monetized benefits of air pollution abatement. The relationship between mortality and ambient PM concentration is well established, while this is not the case for other pollutants. Moreover, there is some evidence that there are synergistic effects between PM and other pollutants (e.g. ozone). Therefore it is desirable to transfer estimates from studies that consider multiple pollutants as explanatory variables in regression models.

Quantifying Mortality due to Particulate Matter

In epidemiological studies of PM, typical measurable health endpoints include all-cause and cause specific mortality, as well as hospital admissions and emergency room visits. Tables 3.3 and 3.4 summarize relative risk estimates associated with PM pollution from the four available prospective cohort studies. The Pope et al. (2002) study estimates relative risk associated with a 10 $\mu g/m^3$ increase in $PM_{2.5}$ (particulates less than 2.5 μm in diameter), while the HEI (2000) study consider a 25 $\mu g/m^3$ change. Therefore, the relative risk estimates in these tables are not comparable. At the end of this chapter, in Tables 3.13-3.45 we list dose-response functions from available studies, and for each relative risk estimate we derived an estimate for β. The value of $100\times\beta$ can be interpreted as the percentage change in the health effect associated with a unit increase in the pollutant.

Table 3.3: Pope et al. (2002) estimates of adjusted relative risk (RR) associated with a 10 μg/m³ change in fine particles measuring less than 2.5 μm in diameter

Cause of Mortality	Adjusted relative risk (95% confidence interval)		
	1979-1983	1999-2000	Average
All causes	1.04 (1.01-1.08)	1.06 (1.02-1.10)	1.06 (1.02-1.11)
Cardiopulmonary	1.06 (1.02-1.10)	1.08 (1.02-1.14)	1.09 (1.03-1.16)
Lung cancer	1.08 (1.01-1.16)	1.13 (1.04-1.22)	1.14 (1.04-1.23)
All other causes	1.01 (0.97-1.05)	1.01 (0.97-1.06)	1.01 (0.95-1.06)

Source: Pope et al. (2002), Table 2
Relative risk is adjusted for age, sex, race, smoking, education, body mass, alcohol consumption, occupational exposure, and diet.

Table 3.4: Relative Risks of Mortality Associated with a 24.5 μg/m³ Increase in Fine Particles Using Alternative Measures of PM in the Reanalysis of the Pope et al. (1995) study.

Cause of Mortality	Adjusted relative risk (95% confidence interval)		
	Median $PM_{2.5}$ Pope et al. (1995) measure	Median $PM_{2.5}$ HEI (2000) measure	Mean $PM_{2.5}$ Pope et al. (2000) measure
All cause	1.18 (1.09–1.26)	1.14 (1.06–1.22)	1.12 (1.06–1.19)
Cardiopulmonary	1.30 (1.17–1.44)	1.26 (1.14–1.39)	1.26 (1.16–1.38)
Lung cancer	1.00 (0.79–1.28)	1.08 (0.88–1.32)	1.08 (0.88–1.32)

Source: HEI (2000), Summary Table 4, p.21
Relative risks were calculated for a change in the pollutant of interest equal to the difference in mean concentrations between the most-polluted city and the least-polluted city. In the Pope et al. (1995) sudy, this difference for fine particles was 24.5 μg/m³.

HEI (2000) tested whether the relationship between ambient concentrations/exposure and mortality was linear using the data of Pope et al. Support for both linear and nonlinear relationships was found, depending upon the analytic technique used. Both Pope et al. (1995) and Dockery et al. (1993) used the Cox proportional hazard regression model, under which hazard functions for mortality at two pollutant levels are proportional and invariant in time.

HEI (2000) evaluate the applicability of the Cox proportional hazard model, using flexible concentration-response models, for the data used in Dockery et al. (1993) and Pope et al. (1995). The small number of study locations in Dockery et al. (1993) afforded only a limited opportunity to define the shape of the CR-function. No evidence was found against the linearity of the relationship for PM. For sulfate particles, however, there was some evidence of departures from linearity at both low and high sulfate concentrations. A similar analysis of the Pope et al. (1995) data yielded some evidence of departure from linearity for both PM and sulfate particles. Overall, however, the Cox proportional hazards assumption did not appear inappropriate. Finally, Pope et al. (2002) conclude that within the range of pollution observed in the study, the CR relationship appears to be monotonic and nearly linear.

Quantifying Mortality due to Ozone

There is considerable epidemiologic evidence concerning the relationship between ambient ozone concentrations and human mortality risks. Because ozone contributes to acute (short-term) health effects, the association between daily ozone concentrations and daily mortality is of primary interest to researchers. Table 3.5 below summarizes the findings of a number of studies that found a statistically significant relationship between daily mortality and daily ozone concentrations. These studies show mixed findings as to whether there is a statistically significant association between daily ozone concentrations and daily mortality in each of the study areas.

Table 3.5 Studies for daily mortality and ambient ozone				
Study	***Study Location/Duration***	***Co-pollutants in the model***	***O_3 concentration measure (ppb)***	***Relative risk and 95% confidence interval for a 25 ppb increase in O_3***
Ito and Thurston (1996)	Cook County, Illinois 1985-1990	PM_{10}	Average same day and previous day 1-hr maxima	1.016 (1.004-1.029)
Kinney et al. (1995)	Los Angeles County 1985-1990	PM_{10}	Daily 1-hour maximum	1.000 (0.089-1.010)
Verhoeff et al. (1996)	Amsterdam, Netherlands 1986-1992	PM_{10} Black smoke	Daily 1-hour maximum (2-day lag)	with PM_{10} 1.024 (0.974 -1.078) with black smoke 1.014 (0.984 - 1.046)
Anderson et al. (1997)	London, England 1987-1992	Black smoke	8-hour average and daily 1-hour max (1-day lag)	1.029 (1.015 - 1.042)
Kwon et al. (2001)[2]	Seoul, South Korea 1994-1998	TSP, SO_2, NO_2, CO, O_3	8-hour average	1.01 (1.002-1.017)
Goldberg et al. (2001)	Montreal, Quebec 1984-1993	O_3, SO_2, NO_2, CO, PM_{10}, $PM_{2.5}$	Daily average, 3-day running mean	1.033 (1.017-1.05)
Hong et al. (2002)[3]	Seoul, South Korea 1991-1997	TSP, SO_2, Lagged NO_2, CO, O_3	8-hour average	1.06 (1.02-1.10)
Moolgavkar et al. (1995)	Philadelphia 1973-1988	TSP, SO_2	Daily average	1.015 (1.004-1.026)
Samet et al. (1997)	Philadelphia 1973-1988	TSP, SO_2, NO_2 Lagged CO	2-day average	1.024 (1.008-1.039)

Notes:
1. O_3 was measured in $\mu g/m^3$. To convert to ppb, ozone concentrations in $\mu g/m^3$ were divided by 1.96. In general, the conversion factor depends on the temperature in the study area.
2. The effect of air pollution on daily mortality of patients with congestive heart failure was studied.
3. Only ischemic stroke mortality was included in the study.

Quantifying Mortality due to Carbon Monoxide

A number of studies examined the relationship between daily mortality and concentrations of CO. Cardiovascular mortality was found strongly associated with CO concentrations. The table below summarizes the results and characteristics of several studies.

Table 3.6
Studies for Carbon Monoxide and Mortality

Study	***Location, Study Period and Population***	***Pollutants***	***Endpoints***	*Main Findings*	*Comment*
Burnett et al. (1998)	Toronto, Canada 1980-1984 All ages	CO, NO_2, SO_2, O_3, SO_4, TSP, COH, PM_{10}, $PM_{2.5}$	Non-accidental mortality	Significant effect found in all two-pollutant models. Controlling for CO, significant effect found for SO_4, TSP, COH, PM_{10} and $PM_{2.5}$	Association with cardiac-related mortality is stronger, but CO is also significantly related to non-cardiac mortality. PM_{10} and $PM_{2.5}$ estimated from SO_4, TSP and COH
Kinney et al. (1995)	Los Angeles County 1985-1990 All ages	CO, O_3, PM_{10}	Non-accidental mortality	In single pollutant model, CO is significant, and PM_{10} and O_3 are marginally significant. In the model with CO and PM_{10}, both CO and PM_{10} are significant.	Magnitude of single-pollutant CO relationship drops modestly with the inclusion of PM_{10}.
Saldiva et al. (1995)	São Paulo, Brazil 1990-91 Elderly (65+ years)	CO, O_3, PM_{10}, SO_2, NO_x	Mortality from natural causes	CO significant in single-pollutant model, not significant in any multi-pollutant model.	
Touloumi et al. (1996)	Athens, Greece 1987-1991 All ages	CO, SO_2, black smoke	Total mortality	CO, SO_2, and black smoke significant in single pollutant models	Deaths during one month summertime heat wave were excluded from the analysis

Table 3.6
Studies for Carbon Monoxide and Mortality (continued)

Study	*Location, Study Period and Population*	*Pollutants*	*Endpoints*	*Main Findings*	*Comment*
Mar et al. (2000)	Phoenix, Arizona 1995-1997	$PM_{2.5}$, PM_{10}, SO_2, NO_2, CO	Cardiovascular mortality	Cardiovascular mortality was significantly associated with CO, NO_2, SO_2, $PM_{2.5}$, PM_{10}, PM_{CF} and elemental carbon.	Both combustion-related pollutants and secondary aerosols (sulfates) were associated with cardiovascular mortality.
Hong et al. (2002)	Seoul, South Korea 1991-1997	TSP, SO_2, Lagged NO_2, CO, O_3	Ischemic Stroke Mortality (ICD9:431,434; ICD10: 161,163)	Significantly increased relative risks were found for same-day TSP and SO_2, for NO_2 and CO with a 1-day lag, and for O_3 with a 3-day lag for each interquartile range increase in the pollutant concentration.	

Quantifying Morbidity Benefits

Quantifying morbidity benefits is more difficult for chronic (long-term) conditions than for acute (short-term) health effects, because it requires data on exposure over a long period of time. The most frequently used endpoints in the epidemiologic literature are hospital admissions for various respiratory and cardiovascular illnesses, emergency department visits, chronic diseases (e.g. chronic bronchitis), and minor health effects, such as upper respiratory symptoms (URS), lower respiratory symptoms (LRS), asthma attacks, shortness of breath, work loss days, minor restricted activity days, etc.

Table 3.7 below briefly describes the available studies that found an association between chronic illness and air pollution. Table 3.8 lists the available studies for minor illness, and Tables 3.9 and 3.10 summarize studies of hospital admissions for respiratory and cardiovascular causes, respectively.

Table 3.7
Studies for Chronic Illness and Air Pollution

Study	*Location, Study Period and Population*	*Pollutants*	*Endpoints*	*Main Findings*	*Comment*
Portney and Mullahy (1990)	Nationwide Sample form the 1979 National Health Interview Survey 1,318 persons age 17-93	O_3, TSP	Sinusitis, hay fever, AOD	Controlling for TSP, O_3 significantly related to the initiation (or exacerbation) of sinusitis and hay fever; no effect on AOD. TSP not significantly related to any endpoint, although it is marginally significant for AOD.	
Schwartz (1993)	Nationwide sample from the National Health and Nutrition Examination Survey 1974-75 6,138 individuals ages 30-74	TSP, SO_2	Chronic bronchitis, asthma, shortness of breath (dyspnea), respiratory illness	TSP significantly related to the prevalence of chronic bronchitis, and marginally significant for respiratory illness. No effect on asthma or dyspnea.	Respiratory illness defined as a significant condition, coded by an examining physician as ICD8 code (460-519)
Xu et al. (1993)	Beijing, China, Survey conducted August-September 1986 1,576 never-smokers	TSP, SO_2	Chronic bronchitis, asthma	Chronic bronchitis significantly higher in the community with the highest TSP level. TSP not linked to the prevalence of asthma.	
Zemp et al. (1999)	Eight sites in Switzerland 1991 9,651 individuals ages 18-60	TSP, PM_{10}, NO_2, O_3	Chronic phlegm, chronic cough, breathless-ness, asthma, dyspnea in exertion	Single-pollutant models: PM_{10} and NO_2 significantly associated with chronic cough or phlegm, breathlessness and dyspnea. Similar though less significant associations found for TSP. No significant effect found for O_3.	

Table 3.7
Studies for Chronic Illness and Air Pollution (continued)

Study	*Location, Study Period and Population*	*Pollutants*	*Endpoints*	*Main Findings*	*Comment*
Abbey et al. (1993)	California initial survey: 1977 final survey: 1987 3,914 Seventh Day Adventists	TSP, O_3, SO_2	AOD, chronic bronchitis, asthma	TSP linked to new cases of AOD and chronic bronchitis, but not to asthma or the severity of asthma. O_3 not linked to the incidence of new cases of any endpoint, but O_3 was linked to the severity of asthma. No effect found for SO_2.	Emphysema, chronic bronchitis, and asthma comprise AOD
Abbey et al. (1995)	California initial survey: 1977 final survey: 1987 1,868 Seventh Day Adventists	$PM_{2.5}$	AOD, chronic bronchitis, asthma	$PM_{2.5}$ related to new cases of chronic bronchitis, but not to new cases of AOD or asthma	$PM_{2.5}$ estimated from visibility data
Chapman et al. (1985)	4 Utah communities 1976 5.623 young adults	SO_2, SO_4, NO_3, TSP	Persistent cough and phlegm	Persistent cough and phlegm is higher in the community with higher SO_2, SO_4 and TSP concentrations	
McDonnell et al. (1999)	California initial survey: 1997 final survey: 1992 3,091 Seventh Day Adventists	O_3, PM_{10}, SO_2, SO_4, NO_2	Asthma	Single-pollutant models: O_3 significantly linked to asthma cases in males, but not females; other pollutants not significantly linked to new asthma cases in males or females. Two-pollutant models estimated for ozone with another pollutant; little impact found on size of ozone coefficient	Average pollution level from 1973-1992 used. Prior to 1987, PM_{10} from TSP.

Table 3.8
Studies for Minor Illness

Endpoints	*Study Population*	*Study*	*Pollutants Used in Final Model*
Acute bronchitis	Ages 8-12	Dockery et al. (1996)	$PM_{2.5}$
Upper respiratory symptoms (URS)	Ages 9-11	Pope et al. (1991)	PM_{10}
Lower respiratory symptoms (LRS)	Ages 7-14	Schwartz et al. (1994)	$PM_{2.5}$
Respiratory illness	Ages 6-7	Hasselblad et al. (1992)	NO_2
Any of 19 respiratory symptoms	Ages 18-65	Krupnick et al. (1990)	O_3, PM_{10}
Moderate or worse asthma	All ages (asthmatics)	Ostro et al. (1991)	$PM_{2.5}$
Asthma attacks	All ages (asthmatics)	Whittemore and Korn (1980)	O_3, PM_{10}
Chest tightness, shortness of breath, or wheeze	All ages (asthmatics)	Linn et al. (1987,1988,1990) and Roger and al. (1985)	$PM_{2.5}$
Shortness of breath	Ages 7-12 (African American asthmatics)	Ostro et al. (1991)	$PM_{2.5}$
Work loss days	Ages 18-65	Ostro (1987)	PM_{10}
Minor restricted activity days (MRAD)	Ages 18-65	Ostro and Rothschild (1989)	$PM_{2.5}$, O_3
Restricted activity days	Ages 18-65	Ostro (1987)	$PM_{2.5}$

Table 3.9 Description of Hospital Admissions Studies for Respiratory Illnesses

Study	*Location and period*	*Sample Population*	*Endpoint*	*Pollutants*	*Main Findings*
Schwartz (1994a)	Detroit, MI, 1986-1989	Ages 64+	asthma (493), pneumonia (480-486), non-asthma COPD (491-492, 494-496)	O3, PM10	PM 10 significant and O3 not significant for btoh pneumonia and COPD admissions in single-pollutant models.
Schwartz (1994b)	Minneapolis, MN, 1986-1989	Ages 64+	asthma (493), pneumonia (480-487), non-asthma COPD (491-492, 494-496)	O3, PM10	No association found for asthma admissions. Both O3 and PM10 are significant for pneumonia and COPD admissions.
Schwartz (1994c)	Birmingham, AL, 1986, 1989	Ages 64+	pneumonia (480-487), COPD (490-496)	O3, PM10	PM10 marginally significant for pneumonia admissions. O3 significant for COPD admissions. Both pollutants are significant for all respiratory admissions.
Burnett et al. (1997)	Toronto, Canada, 1992-1994	All ages	all respiratory (464-466,480-486,490-494,496)	CO, O3, PM2.5, PM2.5-10, PM10, SO2	O3 linked to respiratory admissions, PM less strongly linked. CO, NO2, and SO2 are not significant in two-pollutant models.

Table 3.9 Description of Hospital Admissions Studies for Respiratory Illnesses (continued)

Study	*Location and period*	*Sample Population*	*Endpoint (ICD9 codes)*	*Pollutants*	*Main Findings*
Moolgavkar et al. (1997)	Minneapolis, MN, Birmingham, AL, 1986-1991	Ages 64+	pneumonia (480-487), COPD (490-496), respiratory (480-487, 490-496)	CO, O3, PM2.5, PM2.5-10, PM10, SO2	O3 significant in multi-pollutant model for pneumonia admissions. No significant effect found for COPD admissions.
Burnett et al. (1999)	Toronto, Canada, 1980-1994	All ages	asthma (493), respiratory infection (464,466,480-487,494),COPD, (490-492,496)	CO, O3, PM2.5, PM2.5-10, PM10, SO2	O3, CO, PM2.5-10 significant for asthma admissions. O3, NO2, PM2.5 chosen in stepwise regression for respiratory infection. O3 and PM2.5-10 significant for COPD admissions.
Sheppard et al. (1999)	Seattle, WA, 1987-1994	Ages 65+	Asthma (493)	CO, O3, PM2.5, PM2.5-10, PM10, SO2	In multi-pollutant models PM2.5 and CO are most significantly related to asthma admissions.
Linn et al. (2000)	South Coast Air Basin, CA, 1992-1995	Ages 30+	asthma (493), COPD	CO, O3, PM10	Pulmonary disease associated more with NO2 and PM10 than CO. High O3 concentrations seem to present less risk.

Table 3.9 Description of Hospital Admissions Studies for Respiratory Illnesses (continued)

Study	*Location and period*	*Sample Population*	*Endpoint*	*Pollutants*	*Main Findings*
Moolgavkar (2000)	Cook County, IL, Los Angeles County, CA, Maricopa County, AZ, 1987-1995	Ages 65+	COPD (490-496)	CO, O3, PM2.5, PM10, SO2	In Cook and Maricopa counties there was a weak association between admissions and O3, and no association with PM10. In Los Angeles gaseous pollutants other than ozone were strongly associated with COPD admissions. PM was significant only in single-pollutant models.
Zanobetti et al. (2000)	Chicago, Cook County, IL, 1985-1994	All ages	COPD (490-496 except 493), pneumonia (480-487), asthma (493), acute bronchitis (466), acute respiratory illness (460-466)	PM10	Diagnosis of conduction disorders or dysrhythmias increased the associations between hospital admissions for COPD and pneumonia and PM10. Persons with asthma had twice the risk of PM10-related pneumonia admissions, and persons with heart failure had twice the risk of PM10-induced COPD admissions
Lin et al. (2003)	Toronto, Canada, 1981-1993	Ages 6-12	asthma (493)	CO, O3, PM2.5, PM2.5-10, PM10, SO2	Significant acute effect of CO was found in boys, and SO2 showed significant effects of chronic exposure in girls. NO2 was positively associated with admissions in both sexes. O3 was not significant.
Chen et al. (2004)	Vancouver, Canada, 1995-1999	Ages 65+	COPD (490-492, 494, 496)	CO, O3, PM2.5, PM2.5-10, PM10, SO2	PM measures had a positive association with hospital admissions for COPD. The associations were no longer significant when NO2 was included in the models.

Table 3.10 Description of Hospital Admissions Studies for Cardiovascular Illnesses

Study	*Location and period*	*Sample Population*	*Endpoint (ICD9 codes)*	*Pollutants*	*Main Findings*
Schwartz and Morris (1995)	Detroit, MI, 1986-1989	Ages 64+	ischemic heart disease (410-414), dysrhythmias (427), congestive heart failure (428)	CO, O3, PM10, SO2	PM10 and Co significant in two-pollutant models for ischemic heart disease. No significant effect found for dysrhythmias. In two-pollutant models PM10 is significant for congestive heart failure admissions.
Burnett et al. (1997)	Toronto, Canada, 1992-1994	All ages	cardiac (410-414, 427-428)	CO, O3, PM2.5, PM2.5-10, PM10, SO2	Significant affect found for O3, and to a lesser extent, for PM. Other pollutants are not significant.
Schwartz(1997)	Tucson AZ, 1988-1990	Ages 64+	cardiovascular disease (390-429)	CO, O3, PM10, SO2	CO and PM10 significant in two-pollutant models. No effect seen for other pollutants.
Burnett et al. (1999)	Toronto, Canada, 1980-1994	All ages	ischemic heart disease (410-414), dysrhythmias (427), congestive heart failure (428)	CO, O3, PM2.5, PM2.5-10, PM10, SO2	NO2 and SO2 significant for ischemic heart disease. O3, CO, and PM2.5 significant for dysrhythmias. NO2 and CO significant for congestive heart failure.

Table 3.10 Description of Hospital Admissions Studies for Cardiovascular Illnesses (continued)

Study	*Location and period*	*Sample Population*	Endpoint (ICD9 codes)	Pollutants	Main Findings
Schwartz (1999)	8 U.S. counties, 1988-1990	Ages 65+	cardiovascular disease (390-429)	CO, PM10	Both pollutants significant in two-pollutant models
Linn et al.(2000)	South Coast Air Basin, CA, 1992-1995	Ages 30+	congestive heart failure, myocardial infection, cardiac arrhythmia	CO, O3, PM10	CO, NO2, and to a lesser extent PM10 showed consistently significant relationship to cardiovascular admissions. O3 was negatively associated or not significant.
Zanobetti et al. (2000)	Chicago, Cook County, IL, 1985-1994	All ages	cardiovascular disease (390-429), myocardial infarction (410), congestive heart failure (428), conduction disorders (426), dysrthythmias (427)	PM10	PM10-related hospital admissions for cardiovascular diseases were almost doubled in subjects with concurrent respiratory infections.
Mann et al. (2002)	South Coast Air Basin, CA, 1988-1995	All ages	ischemic heart disease (410-414)	CO, O3, PM10	CO was associated with same-day IHD admissions in persons with secondary diagnosis of arrhythmia.
Koken et al. (2003)	Denver, Colorado, 1993-1997	Ages 65+	Pulmonary Heart Disease, Coronary Atherosclerosis, Congestive Heart Failure, Cardiac Dysrhythmias	CO, O3, PM10, SO2	SO2 appears to be related to increased hospital stays for cardiac dysrhythmias, and CO is associated with congestive heart failure. No association was found with PM or NO2.

3.3 VALUATION OF HEALTH BENEFITS

3.3.1 Monetizing mortality benefits

Environmental economics developed a number of methods for estimating health benefits from avoided air pollution. The most popular primary methods are described below:

Contingent valuation method *(CVM)* is a survey-based method to determine willingness-to-pay (WTP) for a hypothetical change in environmental effects.

Averting behavior is a method to infer WTP from the costs and effectiveness of actions taken to avoid a negative environmental effect.

Cost-of-illness (COI) or ***damage costs*** methods involve estimating direct costs (such as, medical expenses) and indirect costs (for example, forgone earnings) of an environmental effect.

Hedonic methods estimate WTP for an environmental amenity by inferring its value from the market price of another (but in some sense related) good. For example, the *hedonic property values* method estimates a marginal WTP function based on an estimated relationship between housing prices and housing attributes (that include environmental amenities, such as good visibility or air quality). In contrast, the *hedonic wages* method estimates the value of environmental amenities from a worker's WTA a higher salary to compensate for exposure to higher levels of risk on the job.

Mortality benefits are most often monetized using a Value of Statistical Life (VSL) estimate. VSL is a measure of the WTP for reductions in the risk of premature death aggregated over the population experiencing the potential risk reduction. For example, if each person out of one million people is willing to pay five dollars for a 1:1,000,000 reduction in mortality risk, then on average one life is saved, and hence the value of VSL is $5,000,000. (EPA relies on a composite VSL estimate based on 26 VSL studies – 21 of which use the hedonic wage method, and 5 use CVM).

Despite its widespread use, the VSL concept has been subject to criticism. One shortcoming of the VSL is highlighted when one considers the difference between mortality due to acute (short-term) and mortality due to chronic (long-term) exposure. One of the basic assumptions underlying the VSL approach is that that equal increments in fatality risk are valued equally irrespective of the initial risk. This assumption is defensible only if the prior risk is small. Some experts have suggested that it is not appropriate to estimate mortality that is the result of acute exposure, because people affected usually have a pre-existing disease and a relatively high prior risk of mortality.

Alternative measures to VSL include the Value of Statistical Life Years (VSLY) lost or saved. For example, if pollution abatement saves one person with average life expectancy of fifty more years, then we say that the policy results in fifty life years extended. VSLY

can be interpreted as an age-specific VSL. Another alternative to VSL is the Quality Adjusted Life Years (QALY) measure. QALY adjusts life-years extended for the quality of life during those years. To estimate QALY, we need information about the path and duration of health states, as well as we have to choose weights for the different health states.

The primary approach of estimating VSL has been the use of hedonic wage and hedonic price models that examine the equilibrium risk choices. The observed market decisions (measured by wages and prices) reflect the joint influence of supply and demand in the market. Most of the empirical literature has concentrated on valuing mortality risk by estimating compensating differentials for on-the-job risk exposure in labor markets. Viscusi and Aldy (2003) provide a meta-analysis of the extensive literature of VSL based on estimates using U.S. labor market data from the last three decades. The main results of these studies are summarized in Table 3.11. Estimates of VSL typically range from $4 million to $9 million.

The wage-risk relationship is typically estimated by regressing the observed wage of individuals on a vector of personal characteristics, a vector of job characteristics, fatality and non-fatality risk associated with the job, and the workers' compensation benefits payable for a job injury suffered by the worker. An important methodological question is the choice of a risk measure. An ideal risk measure would reflect both the employee's perception of on-the-job fatality risk and the employer's perception of such risk. Suitable measures of the subjective risk are rarely available, and therefore the standard approach in the literature has been the use of industry-specific or occupation-specific risk measures (e.g. average number or rate of fatalities over a period of several years).

Hedonic wage studies vary in several aspects, such as, their labor market coverage (entire labor force vs. specific occupations), geographic coverage (entire country vs. specific states or regions), class of workers (e.g. blue-collar workers only), gender, union-status of workers (e.g. union-members only), as well as, the measure of mortality risk used.

Table 3.11 Summary of Labor Market Studies of the Value of a Statistical Life, United States					
Author (Year)	***Sample***	***Risk Variable***	***Mean Risk***	***Average Income Level (2000 US$)***	***Implicit VSL (millions 2000 US$)***
Smith (1974)	Current Population Survey (CPS) 1967 Census of Manufactures 1963 U.S. Census 1960 Employment and Earnings 1963	Bureau of Labor Statistics (BLS) 1966 1967	0.000125	$29,029	$9.2
Thaler and Rosen (1975)	Survey of Economic Opportunity 1967	Society of Actuaries 1967	0.001	$34,663	$1.0
Smith (1976)	CPS 1967 1973	BLS 1966 1967 1970	0.0001	$31,027	$5.9
Viscusi (1978a 1979)	Survey of Working Conditions 1969-1970 (SWC)	BLS 1969 subjective risk of job (SWC)	0.0001	$31,842	$5.3
Brown (1980)	National Longitudinal Survey of Young Men 1966-71 1973	Society of Actuaries 1967	0.002	$49,019	$1.9
Viscusi (1981)	Panel Study of Income Dynamics (PSID) 1976	BLS 1973-1976	0.0001	$22,618	$8.3
Olson (1981)	CPS 1978	BLS 1973	0.0001	$36,151	$6.7
Arnould and Nichols (1983)	U.S. Census 1970	Society of Actuaries 1967	0.001	NA	$0.5-$1.3
Butler (1983)	S.C. Workers' Compensation Data 1940-69	S.C. Workers' Compensation Claims Data	0.00005	$22,713	$1.3

Table 3.11 Summary of Labor Market Studies of the Value of a Statistical Life, United States (continued)

Author (Year)	*Sample*	*Risk Variable*	*Mean Risk*	*Average Income Level (2000 US$)*	*Implicit VSL (millions 2000 US$)*
Low and McPheters (1983)	International City Management Association 1976 (police officer wages)	Constructed a risk measure from DOJ/FBI police officers killed data 1972-75 for 72 cities	0.0003	$33,172	$1.4
Dorsey and Walzer (1983)	CPS May 1978	BLS 1976	0.000052	$21,636	$11.8- $12.3
Leigh and Folsom (1984)	PSID 1974 Quality of Employment Survey (QES) 1977	BLS	0.0001	$29,038 $36,946	$10.1-$13.3
Smith and Gilbert (1984 1985)	CPS 1978	BLS 1975	NA	NA	$0.9
Dillingham and Smith (1984)	CPS May 1979 BLS industry data 1976 1979	NY Workers' Comp Data 1970	0.000082	$29,707	$4.1-$8.3
Dillingham (1985)	QES 1977	BLS 1976 NY Workers' Compensation data 1970	0.000008 0.00014	$26,731	$1.2 $3.2-$6.8
Leigh (1987)	QES 1977 CPS 1977	BLS	NA	NA	$13.3
Moore and Viscusi (1988a)	PSID 1982 BLS 1972-1982	NIOSH National Traumatic Occupational Fatality (NTOF) Survey 1980-1985	0.00005 0.00008	$24,931	$3.2 $9.4

Source: Viscusi and Aldy (2003), Table 2, p.88

Half of the studies reviewed in Viscusi and Aldy (2003) provide estimates that range form $5 million to $12 million (in 2000 dollars). Estimates below $5 million usually are reported by studies that use the Society of Actuaries data, which contains data on workers that self-selected themselves to jobs that are much riskier than average. On the other hand, studies that report estimates above $12 million tend to estimate the wage-risk relationship indirectly. Viscusi and Aldy (2003) regard the median estimate of $7 million from the above table as the most reliable.

These values of VSL using hedonic wage methods are similar to those generated by U.S. product market and housing market studies.

When transferring the estimates of VSL to non-labor market contexts, as is the case of cost-benefit analysis of the RPS, we must make sure that the preferences of the study population and the populations in the policy context are similar. Other factors that may

influence the transfer of VSL estimates are the age and income distribution of the study population. In addition to finding a positive association between income and VSL, Viscusi and Aldy (2003) also found a statistically significant relationship between union-status of workers and VSL.

3.3.2 Valuation of Morbidity Benefits

There are a number of health effects (endpoints) that can be quantified (U.S. EPA, 1989), but difficult to value in monetary terms. These effects include, for example, reduced lung function. Currently, there are no studies available on the economic valuation of changes in lung function. One reason is that there is no clear connection between lung function and the economic well-being of an individual. Reduced lung function is typically associated with other symptoms, such as cough or asthma attacks, and it is unclear whether a temporary decrement in lung function would go unnoticed without the related symptoms, and hence it may have no economic value.

Several endpoints reported in the literature overlap with each other, for example the various measures of restricted activity, or their definitions are not unique. Therefore, one must be careful not to include a combination of endpoints that could lead to double counting of benefits.

In a number of studies, it has been found that the different methods, mentioned in the previous sections, generate systematically different estimates for morbidity effects. The following table contains estimates of WTP and cost-of-illness for various health effects. For each effect, both estimates are from the same source. We can conclude from these results, that people's WTP for avoiding certain symptoms usually exceeds the cost of that symptom by an order of a magnitude. On the other hand there does not seem to be a systematic relationship between the two estimates.

Table 3.12 Comparison of the value of morbidity effects using different valuation methods

Symptoms	WTP method	WTP $1996	Individual COI $1996
Berger et al. (1987) Dollar value for one symptom day			
Cough	CVM	$114.74	$18.38
Sinus Congestion	CVM	$41.26	$10.25
Throat Congestion	CVM	$66.34	$21.55
Itchy Eyes	CVM	$73.21	$21.99
Heavy Drowsiness	CVM	$214.44	$2.72
Headache	CVM	$164.16	$5.21
Nausea	CVM	$72.30	$3.78
All Symptoms	CVM	$121.76	$5.93
Chestnut et al. (1988, 1996) Dollar value for one episode			
Symptoms	WTP method	WTP $1996	Individual COI $1996
Angina Episodes	AB	$54.40	$18.54
Angina Episodes	CVM	$57.26	$18.54
Angina Episodes	CVM	$60.13	$18.54
Angina Episodes	CVM	$147.45	$18.54
Dickie-Gerking (1991) Dollar value for a reduction to zero days/year of ozone			
Health effects of ozone for concentration	WTP method	WTP $1996	Medical expenses $1996
Over 12 pphm	AB	$138.53	$36.45
Over 12 pphm	AB	$167.69	$84.57
Over 12 pphm	AB	$249.35	$67.08
Over 12 pphm	AB	$304.76	$160.40
Over 9 pphm	AB	$249.35	$59.79
Over 9 pphm	AB	$298.93	$131.24
Over 9 pphm	AB	$380.58	$94.78
Over 9 pphm	AB	$457.87	$215.81
Rowe-Chestnut (1985)			
Asthma Severity	WTP method	WTP $1996	$1996 Medical expenses	Foregone Earnings $1996
Asthma Severity	CVM	$ 631.70	$196.91	$116.57
Asthma Severity	CVM	$ 919.98	$196.91	$116.57
Asthma Severity	CVM	$ 697.86	$70.89	$116.57

Source: U.S. EPA (2000)

Valuation of hospital admissions avoided

The typical approach for valuing the avoided incidence of hospital admissions is through the use of COI method (U.S. EPA, 1999). Well-developed and detailed estimates of hospitalization of the health effects are readily available (U.S. EPA, 2004). COI estimates should be obtained for each health effects for which dose-response functions are available. Valuation estimates typically have two components: cost of hospital stay, and lost earnings due to hospitalization. As mentioned above, COI method underestimates WTP by a factor of at least three. However, there are currently no studies available that would estimate WTP directly.

CONCENTRATION-RESPONSE FUNCTIONS FOR OZONE (O_3)

Table 3.13 CR-Functions for Ozone (O_3)
Health Endpoint: Onset of asthma

CR-function: $$\Delta\text{chronic asthma} = -\left[\frac{y_1}{(1-y_1)\cdot e^{\beta\Delta O_3} + y_1} - y_1\right]\cdot \text{pop}$$

y_1 = occurrence rate of chronic asthma
β = O_3 coefficient
ΔO_3 = change in O_3 concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	Ozone Concentration Measure	β^1
McDonnell et al. (1999)	California	Non-asthmatic males ages 27+	None	Annual average 8-hour O_3	0.0277 (0.0135)
McConnell et al. (2002)	Southern California	Children ages 9-16	PM_{10}, NO_2	Annual 10:00h to 18:00h mean ozone concentration	0.0904 (0.0213)

Notes: 1 Standard error of β in brackets.

Table 3.14 CR-Functions for Ozone (O_3)
Health Endpoint: Hospital admissions – pneumonia

CR-function: $$\Delta\text{pneumonia admissions} = -\left[y_1\cdot(e^{\beta\cdot\Delta O_3} - 1)\right]\cdot \text{pop}$$

y_1 = daily admissions rate for pneumonia per person
β = O_3 coefficient
ΔO_3 = change in O_3 concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	Ozone Concentration Measure	β^1
Moolgavkar et al. (1997)	Minneapolis, MN	Population ages 65+	SO_2, NO_2, PM_{10}	Daily average O_3 concentration	0.00370 (0.00103
Schwartz (1994b)	Detroit, MI	Population ages 65+	PM_{10}	Daily average O_3 concentration	0.00521 (0.0013)
Schwartz (1994c)	Minneapolis, MN	Population ages 65+	PM_{10}	Daily average O_3 concentration	0.00280 (0.00071)

Notes: 1. Standard error of β in brackets.

Table 3.15 CR-Functions for Ozone (O_3)

Health Endpoint: Mortality

CR-function: $\Delta\text{mortality} = -\left[y_1 \cdot (e^{\beta \cdot \Delta O_3} - 1) \right] \cdot \text{pop}$

y_1 = non-accidental deaths per person of any age
β = O_3 coefficient
ΔO_3 = change in O_3 concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	Ozone Concentration Measure	β^1
Kinney et al. (1995)	Los Angeles, CA	Population of all ages	PM_{10}	Daily one-hour maximum O_3	0.00010 (0.000214)
Moolgavkar et al. (1995)	Philadelphia, PA	Population of all ages	SO_2, TSP	Daily average ozone	0.000611 (0.000216)
Ito and Thurston (1996)	Chicago, IL	Population of all ages	PM_{10}	Daily one-hour maximum O_3	0.00068 (0.00029)
Kelsall et al. (1997)	Philadelphia, PA	Population of all ages	CO, NO_2, SO_2, TSP	Daily average ozone	0.000936 (0.000312)
Golberg et al. (2001)	Montréal, Québec	Population of all ages	CO, NO_2, NO, SO_2, $PM_{2.5}$	Daily average ozone	0.002056 (0.000475)

Notes: 1. Standard error of β in brackets.

Table 3.16 CR-Functions for Ozone (O_3)
Health Endpoint: Emergency room visits for asthma

CR-function: $\Delta\text{asthma-related ER visits} = \frac{\beta}{\text{BasePop}} \Delta O_3 \cdot \text{pop}$

BasePop = baseline population
β = O_3 coefficient
ΔO_3 = change in O_3 concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	Ozone Concentration Measure	β^1
Cody et al. (1992)	Northern New Jersey	Population of all ages	None	Daily five-hour average O_3	0.0203 (0.00717)
Weisel et al. (1992)	Northern New Jersey	Population of all ages	None	Daily five-hour average O_3	0.0443 (0.00723)
Stieb et al. (1996)	New Brunswick, Canada	Population of all ages	None	Daily one-hour maximum O_3	0.0035 (0.0018)

Notes: 1. Standard error of β in brackets.

Table 3.17 CR-Functions for Ozone (O_3)
Health Endpoint: Hospital admissions - all respiratory causes

CR-function: $\Delta \text{all respir. admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta O_3} - 1) \right] \cdot \text{pop}$

y_1 = daily admissions rate for all respiratory causes per person
β = O_3 coefficient
ΔO_3 = change in O_3 concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	Ozone Concentration Measure	β^1
Thurston et al. (1994)	Toronto, Canada	Population of all ages	$PM_{2.5}$	Daily one-hour maximum O_3 concentrations	0.00250 (9.71E-9)
Schwartz (1995)	New Haven, CT	Population ages 65+	PM_{10}	Daily average O_3	0.00265 (0.00140)
Schwartz (1995)	Tacoma, WA	Population ages 65+	PM_{10}	Daily average O_3 concentrations	0.00715 (0.00257)
Burnett et al. (1997)	Toronto, Canada	Population of all ages	$PM_{2.5}$, PM_{10}, NO_2, SO_2	Daily one-hour maximum O_3 concentrations	0.00498 (0.00106)

Notes: 1. Standard error of β in brackets.

Table 3.18 CR-Functions for Ozone (O_3)
Health Endpoint: Hospital admissions for chronic obstructive pulmonary disease (COPD)

CR-function: $\Delta \text{COPD admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta O_3} - 1) \right] \cdot \text{pop}$

y_1 = daily admissions rate for COPD per person
β = O_3 coefficient
ΔO_3 = change in O_3 concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	Ozone Concentration Measure	β^1
Schwartz (1994b)	Detroit, MI	Population 65+	PM_{10}	Daily average O_3 concentration	0.00549 (0.00205)
Moolgavkar et al. (1997)	Minneapolis, MN	Population 65+	CO, NO_2	Daily average O_3 concentration	0.00274 (0.00170)
Burnett et al. (1999)	Toronto, Canada	Population of all ages	CO, $PM_{2.5}$, PM_{10}	Daily average O_3 concentration	0.00303 (0.00110)

Notes: 1. Standard error of β in brackets.

Table 3.19 CR-Functions for Ozone (O_3)
Health Endpoint: Hospital admissions for asthma

CR-function: $\Delta\text{asthma admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta O_3} - 1) \right] \cdot \text{pop}$

y_1 = daily asthma admission rate per person
β = O_3 coefficient
ΔO_3 = change in O_3 concentrations (ppb)
pop = sample population

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	Ozone Concentration Measure	β^1
Burnett et al. (1999)	Toronto, Canada	Population of all ages	CO, $PM_{2.5}$, PM_{10}	Daily average O_3 concentration	0.00250 (0.000718)

Notes: 1. Standard error of β in brackets.

Table 3.20 CR-Functions for Ozone (O_3)
Health Endpoint: Hospital admissions for respiratory infection

CR-function: $\Delta\text{respiratory infections admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta O_3} - 1) \right] \cdot \text{pop}$

y_1 = daily respiratory infections admission rate per person
β = O_3 coefficient
ΔO_3 = change in O_3 concentrations (ppb)
pop = sample population

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	Ozone Concentration Measure	β^1
Burnett et al. (1999)	Toronto, Canada	Population of all ages	$PM_{2.5}$, NO_2	Daily average O_3 concentration	0.00198 (0.000520)

Notes: 1. Standard error of β in brackets.

Table 3.21 CR-Functions for Ozone (O_3)
Health Endpoint: Hospital admissions for cardiac

CR-function: $\Delta\text{cardiac admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta O_3} - 1) \right] \cdot \text{pop}$

y_1 = daily cardiac admission rate per person
β = O_3 coefficient
ΔO_3 = change in O_3 concentrations (ppb)
pop = sample population

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	Ozone Concentration Measure	β^1
Burnett et al. (1997)	Toronto, Canada	Population of all ages	$PM_{2.5}$, PM_{10}	Daily 12-hour average	0.00531 (0.00142)

Notes: 1. Standard error of β in brackets.

Table 3.22 CR-Functions for Ozone (O_3)
Health Endpoint: Hospital admissions for dysrhythmias

CR-function: $\Delta\text{cardiac admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta O_3} - 1) \right] \cdot \text{pop}$

y_1 = daily admission rate for dysrhythmias per person
β = O_3 coefficient
ΔO_3 = change in O_3 concentrations (ppb)
pop = sample population

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	Ozone Concentration Measure	β^1
Burnett et al. (1999)	Toronto, Canada	Population of all ages	CO, $PM_{2.5}$	Daily average O_3	0.00168 (0.00103)

Notes: 1. Standard error of β in brackets.

Table 3.23 CR-Functions for Ozone (O_3)
Health Endpoint: Presence of any of 19 acute respiratory symptoms (ARD)

CR-function: $\Delta ARD2 \cong \beta^*_{PM10} \cdot \Delta O_3 \cdot pop$

β^* = first derivative of the stationary probability
ΔO_3 = change in daily one-hour maximum O_3 concentrations (ppb)
pop = sample population

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	Ozone Concentration Measure	β[1]
Krupnick et al. (1990)	Glendora-Covina-Azusa, CA	Population aged 18-65	SO_2, COH	1-hour maximum	0.000137 (0.0000697)

Notes: 1. Standard error of β in brackets.

Table 3.24 CR-Functions for Ozone (O_3)
Health Endpoint: Other minor health effects

Self-reported asthma attacks	$\Delta\text{asthma attacks} = \left[\frac{y_1}{(1-y_1)\cdot e^{\Delta O_3 \beta} + y_1} - y_1 \right] \cdot \text{pop}$			Whittemore and Korn (1980) Location: Los Angeles, CA Other pollutants: TSP
	y_1	=	daily incidence of asthma attacks = 0.027	
	β	=	O_3 coefficient = 0.00187	
	ΔO_3	=	change in daily one-hour maximum O_3 concentration (ppb)	
	pop	=	population of asthmatics of all ages = 5.61% of the population of all ages	
	σ_β	=	standard error of β = 0.000714	
Respiratory and non-respiratory conditions resulting in minor restricted activity days (MRAD)	$\Delta\text{MRAD} = -\left[y_1 \cdot (e^{\beta \cdot \Delta O_3} - 1) \right] \cdot \text{pop}$			Ostro and Rothschild (1989) Location: U.S. Other pollutants: $PM_{2.5}$
	y_1	=	daily MRAD incidence per person = 0.02137	
	β	=	inverse-variance weighted $PM_{2.5}$ coefficient = 0.00220	
	ΔO_3	=	change 2-week average of the daily 1-hour	
	pop	=	maximum O_3 concentration (ppb) population 18-65	
	σ_β	=	standard error of β = 0.000658	

Source:
Costs and Benefits of the Clean Air Act, 1990-2010, Appendix D, Human Health Effects of Criteria Pollutants, EPA

CONCENTRATION-RESPONSE FUNCTIONS FOR PARTICULATE MATTER (PM)

Table 3.25 CR-Functions for Particulate Matter (PM)
Health Endpoint: Mortality

$$\Delta \text{nonaccidental mortality} = -\left[y_1 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1) \right] \cdot \text{pop}$$

CR-function:

y_1 = non-accidental deaths per person
β = $PM_{2.5}$ coefficient
$\Delta PM_{2.5}$ = change in $PM_{2.5}$ concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Dockery et al. (1993)	6 U.S. cities	Population ages 25+	None	0.0124 (0.00423)
Pope et al. (1995)	50 U.S. cities	Population ages 30+	None	0.006408 (0.001509)
HEI (2000)	6 U.S. cities	Population ages 25+	Air toxics	0.01327 (0.004408)
Pope et al. (2002)	50 U.S. cities	Population ages 30+	Sulfate, SO_2, NO_2, CO, O_3	0.00583 (0.001963)

Notes: 1. Standard error of β in brackets.

Table 3.26 CR-Functions for Particulate Matter (PM)
Health Endpoint: Hospital Admissions – obstructive lung disease

$$\Delta \text{ COPD hospital admissions} = -\left[y_1 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1) \right] \cdot \text{pop}$$

CR-function:

y_1 = hospital admissions for obstructive lung disease per person
β = PM coefficient
ΔPM = change in PM concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	PM Metric/Other Pollutants	β^1
Burnett et al. (1999)	Toronto, Canada	Population of all ages	$PM_{2.5\text{-}10}/O_3$	0.0019 (0.0003)

Table 3.27 CR-Functions for Particulate Matter (PM)
Health Endpoint: Hospital Admissions – chronic obstructive pulmonary disease (COPD)

$$\Delta \text{ COPD hospital admissions} = -\left[y_1 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1) \right] \cdot \text{pop}$$

CR-function:

y_1 = hospital admissions for COPD per person
β = PM coefficient
ΔPM = change in PM concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	PM Metric/Other Pollutants	β[1]
Schwartz (1994a)	Birmingham, AL	Population ages 65+	PM_{10}/None	0.00239 (0.000536)
Schwartz (1994b)	Detroit, MI	Population ages 65+	PM_{10}/O_3	0.00202 (0.00059)
Schwartz (1994c)	Minneapolis, MN	Population ages 65+	PM_{10}/None	0.00451 (0.00138)
Moolgavkar et al. (1997)	Minneapolis, MN	Population ages 65+	$PM_{10}/CO,O_3$	0.00088 (0.000777)
Moolgavkar (2000)	Los Angeles County	Population ages 65+	PM_{10}/None	0.00150 (0.000384)
Zanobetti et al. (2000)	Cook County, IL	Population ages 65+	PM_{10}	0.007603 (0.003069)
Chen et al. (2004)	Vancouver, Canada	Population ages 65+	PM_{10}/None	0.01525 (0.004628)

Notes: 1. Standard error of β in brackets.

Table 3.28 CR-Functions for Particulate Matter (PM)
Health Endpoint: Hospital Admissions – pneumonia

$$\Delta \text{ pneumonia hospital admissions} = -\left[y_1 \cdot (e^{-\beta \cdot \Delta PM_{2.5}} - 1) \right] \cdot \text{pop}$$

CR-function:

y_1 = hospital admissions for pneumonia per person
β = PM coefficient
ΔPM = change in PM concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	PM Metric/Other Pollutants	β^1
Schwartz (1994a)	Birmingham, AL	Population ages 65+	PM_{10}/None	0.00174 (0.000838)
Schwartz (1994b)	Detroit, MI	Population ages 65+	PM_{10}/O_3	0.00115 (0.00039)
Schwartz (1994c)	Minneapolis, MN	Population ages 65+	PM_{10}/O_3	0.00157 (0.000677)
Moolgavkar et al. (1997)	Minneapolis, MN	Population ages 65+	PM_{10}/O_3, NO_2, SO_2	0.000498 (0.000505)
Zanobetti et al. (2000)	10 U.S. cities	Population ages 65+	PM_{10}/O_3, SO_2, CO	0.00193 (0.00039)

Notes: 1. Standard error of β in brackets.

CONCENTRATION-RESPONSE FUNCTIONS FOR CARBON MONOXIDE (CO)

Table 3.29 CR-Functions for Carbon Monoxide (CO)
Health Endpoint: Mortality

CR-function: $\Delta\text{mortality} = -\left[y_1 \cdot (e^{\beta \cdot \Delta CO} - 1) \right]$

y_1 = Non-accidental deaths per person of any age
β = CO coefficient
ΔCO = change in CO concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Saldiva et al. (1995)	São Paulo, Brazil	Ages 65+	None	0.015918 (0.00507)
Touloumi et al. (1996)	Athens, Greece	All ages	SO_2	0.005511 (0.00167)
Burnett et al. (1998)	Toronto, Canada	All ages	TSP	0.0266 (0.0038)
Hong et al. (2002)	Seoul, South Korea	All ages	TSP	0.0890 (0.0256)

Table 3.30 CR-Functions for Carbon Monoxide (CO)

Health Endpoint: Hospital admissions for asthma (ICD9-493)

CR-function: $\Delta\text{asthma admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta CO} - 1) \right] \cdot \text{pop}$

y_1 = daily hospital admissions rate for asthma per person
β = CO coefficient
ΔCO = change in average daily CO concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Burnett et al. (1999)	Toronto, Canada	All ages	$PM_{2.5}$, PM_{10}, O_3	0.0332 (0.00861)
Sheppard (1999)	Seattle, WA	Under age 65	$PM_{2.5}$	0.0528 (0.0185)
Linn et al. (2000)	Los Angeles, CA	Population ages 30+	PM_{10}, O_3, NO_2	0.0280 (0.0100)
Lin et al. (2003)	Toronto, Canada	Boys ages 6-12	NO_2, SO2, O_3	0.1353 (0.0589)

Notes: 1.Standard error of β in brackets. 2.ICD = International Classification of Diseases

Table 3.31 CR-Functions for Carbon Monoxide (CO)

Health Endpoint: Hospital admissions for congestive heart failure

CR-function: $\Delta\text{cong. heart failure admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta CO} - 1) \right] \cdot \text{pop}$

y_1 = daily hospital admissions rate for congestive heart failure per person
β = CO coefficient
ΔCO = change in average daily CO concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Schwartz and Morris (1995)	Detroit, MI	Ages 65+	PM_{10}	0.0170 (0.00468)
Burnett et al. (1999)	Toronto, Canada	All ages	NO_2	0.0340 (0.0163)
Koken et al. (2003)	Denver, CO	Ages 65+	NO_2, O_3, PM_{10}	0.3328 (0.1681)

Notes: 1.Standard error of β in brackets. 2. ICD = International Classification of Diseases

Table 3.32 CR-Functions for Carbon Monoxide (CO)

Health Endpoint: Hospital admissions for cardiovascular disease

CR-function: $\Delta\text{cardiovascular admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta CO} - 1) \right] \cdot \text{pop}$

y_1 = daily hospital admissions rate for cardiovascular disease per person
β = CO coefficient
ΔCO = change in average daily CO concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Schwartz (1997)	Tucson, AZ	Ages 65+	PM_{10}	0.0139 (0.00715)
Schwartz (1999)	8 U.S. counties	Ages 65+	PM_{10}	0.0127 (0.00255)

Notes: 1.Standard error of β in brackets. 2. ICD = International Classification of Diseases

Table 3.33 CR-Functions for Carbon Monoxide (CO)

Health Endpoint: Hospital admissions for obstructive lung disease

CR-function: $\Delta\text{obst. lung disease admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta CO} - 1) \right] \cdot \text{pop}$

y_1 = daily hospital admissions rate for obstructive lung disease per person
β = CO coefficient
ΔCO = change in average daily CO concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Moolgavkar[2] (1997)	Minneapolis-St. Paul, MN	Population ages 65+	PM_{10}, O_3	0.0573 (0.0329)
Burnett et al. (1999)	Toronto, Canada	Population of all ages	$PM_{2.5}$, PM_{10}, O_3	0.0250 (0.0165)

Notes: 1.Standard error of β in brackets. 2. Health endpoint: COPD

Table 3.34 CR-Functions for Carbon Monoxide (CO)

Health Endpoint: Hospital admissions for dysrhythmias

CR-function: $\Delta\text{dysrhythmias admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta CO} - 1) \right] \cdot \text{pop}$

y_1 = daily hospital admissions rate for dysrhythmias per person
β = CO coefficient
ΔCO = change in average daily CO concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Burnett et al. (1999)	Toronto, Canada	Population of all	$PM_{2.5}$, O_3	0.0573

		ages		(0.0229)
Notes: 1.Standard error of β in brackets. 2. Health endpoint: COPD				

Table 3.35 CR-Functions for Carbon Monoxide (CO)

Health Endpoint: Hospital admissions for obstructive lung disease

CR-function: $\Delta\text{ischemic heart disease admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta CO} - 1) \right] \cdot \text{pop}$

y_1 = daily hospital admissions rate for dysrhythmias per person
β = CO coefficient
ΔCO = change in average daily CO concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Schwartz and Morris (1995)	Detroit, MI	Population ages 65+	PM_{10}	0.000467 (0.000435)

Notes: 1.Standard error of β in brackets. 2. Health endpoint: COPD

CONCENTRATION-RESPONSE FUNCTIONS FOR NITROGEN DIOXIDE (NO_2)

Table 3.36 CR-Functions for Nitrogen Dioxide (NO_2)

Health Endpoint: Hospital admissions for respiratory conditions

CR-function: $\Delta\text{all respiratory admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta NO_2} - 1) \right] \cdot \text{pop}$

y_1 = daily hospital admissions rate for respiratory conditions per person
β = NO_2 coefficient
ΔNO_2 = change in average daily NO_2 concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Burnett et al. (1997)[2]	Toronto, Canada	Population of all ages	$PM_{2.5}$, PM_{10}, O_3	0.00378 (0.00221)
Burnett et al. (1999)[3]	Toronto, Canada	Population of all ages	$PM_{2.5}$, O_3	0.00172 (0.000521)

Notes: 1.Standard error of β in brackets. 2. Health endpoint: all respiratory, NO_2 measure: 12-hour average
3.Health endpoint: respiratory infection

Table 3.37 CR-Functions for Nitrogen Dioxide (NO_2)

Health Endpoint: Hospital admissions for pneumonia

CR-function: $\Delta\text{pneumonia admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta NO_2} - 1) \right] \cdot \text{pop}$

y_1 = daily hospital admissions rate for pneumonia per person
β = NO_2 coefficient
ΔNO_2 = change in average daily NO_2 concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Moolgavkar (1997)	Minneapolis-St. Paul, MN	Population ages 65+	PM_{10}, O_3, SO_2	0.00172 (0.00125)

Notes: 1.Standard error of β in brackets.

Table 3.38 CR-Functions for Nitrogen Dioxide (NO_2)

Health Endpoint: Hospital admissions for congestive heart failure

CR-function: $\Delta\text{cong. heart failure admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta NO_2} - 1) \right] \cdot \text{pop}$

y_1 = daily hospital admissions rate for congestive heart failure per person
β = NO_2 coefficient
ΔNO_2 = change in average daily NO_2 concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Burnett et al. (1999)	Toronto, Canada	Population of ages	$PM_{2.5}$, O_3	0.00264 (0.000769)

Notes: 1.Standard error of β in brackets.

Table 3.39 CR-Functions for Nitrogen Dioxide (NO_2)

Health Endpoint: Hospital admissions for ischemic heart disease

CR-function: $\Delta\text{ischemic heart disease admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta NO_2} - 1) \right] \cdot \text{pop}$

y_1 = daily hospital admissions rate for ischemic heart disease per person
β = NO_2 coefficient
ΔNO_2 = change in average daily NO_2 concentrations (ppm)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Burnett et al. (1999)	Toronto, Canada	Population of ages	SO_2	0.00318 (0.000521)

Notes: 1.Standard error of β in brackets.

CONCENTRATION-RESPONSE FUNCTIONS FOR SULFUR DIOXIDE (SO_2)

Table 3.40 CR-Functions for Sulfur Dioxide (SO_2)
Health Endpoint: Mortality

CR-function: $\Delta \text{mortality} = -\left[y_1 \cdot (e^{\beta \cdot \Delta SO_2} - 1)\right]$

y_1 = Non-accidental deaths per person of any age
β = SO_2 coefficient
ΔSO_2 = change in SO_2 concentrations (ppb)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Saldiva et al. (1995)	São Paulo, Brazil	Ages 65+	None	0.005204 (0.002229)
Touloumi et al. (1996)	Athens, Greece	All ages	CO	0.00404 (0.00006)
Hong et al. (2002)	Seoul, South Korea	All ages	TSP	0.003343 (0.001126)

Table 3.41 CR-Functions for Sulfur Dioxide (SO_2)
Health Endpoint: Emergency department (ED) admissions for cardiac

CR-function: $\Delta \text{cardiac ED admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta SO_2} - 1)\right]$

y_1 = cardiac ED admissions per person
β = SO_2 coefficient
ΔSO_2 = change in SO_2 concentrations (ppb)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Stieb et al. (2000)	Saint John, Canada	All ages	O_3, PM_{10}, $PM_{2.5}$, SO_4	0.00201 (0.000664)

Table 3.42 CR-Functions for Sulfur Dioxide (SO_2)
Health Endpoint: Emergency department (ED) admissions for respiratory causes

CR-function: $\Delta \text{cardiac ED admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta SO_2} - 1)\right]$

y_1 = respiratory ED admissions per person
β = SO_2 coefficient
ΔSO_2 = change in SO_2 concentrations (ppb)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Stieb et al. (2000)	Saint John, Canada	All ages	O_3, PM_{10}, $PM_{2.5}$, SO_4	0.001527 (0.000460)

Table 3.43 CR-Functions for Sulfur Dioxide (SO_2)
Health Endpoint: Hospital admissions for asthma

CR-function: $\Delta \text{Asthma hospital admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta SO_2} - 1)\right]$

y_1 = hospital admissions for asthma per person
β = SO_2 coefficient
ΔSO_2 = change in SO_2 concentrations (ppb)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Lin et al. (2003)	Toronto, Canada	Children ages 6-12	$PM_{2.5}$	0.035266 (0.012383)

Table 3.44 CR-Functions for Sulfur Dioxide (SO_2)
Health Endpoint: Hospital admissions for COPD

CR-function: $\Delta COPD \text{ hospital admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta SO_2} - 1)\right]$

y_1 = hospital admissions for COPD per person
β = SO_2 coefficient
ΔSO_2 = change in SO_2 concentrations (ppb)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Moolgavkar (2000)	Los Angeles County	Ages 0-19	None	0.0154 (0.0011)
Moolgavkar (2000)	Los Angeles County	Ages 20-64	None	0.0125 (0.0008)
Moolgavkar (2000)	Los Angeles County	Ages 65+	None	0.0113 (0.0010)
Moolgavkar (2000)	Maricopa County	Ages 65+	None	0.0138 (0.0019)

Table 3.45 CR-Functions for Sulfur Dioxide (SO_2)
Health Endpoint: Hospital admissions for ischemic heart disease

CR-function: $\Delta \text{ischemic heart disease hospital admissions} = -\left[y_1 \cdot (e^{\beta \cdot \Delta SO_2} - 1)\right]$

y_1 = hospital admissions for ischemic heart disease per person
β = SO_2 coefficient
ΔSO_2 = change in SO_2 concentrations (ppb)
pop = population sample

Summary of Estimates

Study	Location	Sample Population	Other Pollutants	β^1
Burnett et al. (1999)	Toronto, Canada	All ages	None	0.0009 (0.0001)

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4. INDIRECT BENEFITS TO HUMANS THROUGH ECOSYSTEMS

Air pollution can cause significant ecological damages. Table 4.1 below identifies the most important direct and indirect effects of air pollution.

Table 4.1
Ecological effects of air pollution

Pollutant Class	Major Pollutants and Precursors	Short-term effects	Long-term effects
Acidic Deposition	Sulfuric acid, nitric acid Precursors: SO_2, NO_x	Direct toxic effect to plant leaves and aquatic organisms	Progressive deterioration of soil quality and acidification of surface waters
Nitrogen Deposition	NO_x		Saturation of terrestrial ecosystems with nitrogen. Progressive nitrogen enrichment of coastal estuaries.
Hazardous Air Pollutants (HAPs)	Mercury and dioxins	Direct toxic effects to animals.	Accumulation of mercury and dioxin in the food chain.
Ozone	Tropospheric ozone	Direct toxic effects to plant leaves.	Alteration of ecosystem-wide energy flow and nutrient cycling.
Source: U.S. EPA (1999)			

The first step in valuing ecosystem benefits of reduced air pollution is to identify measurable endpoints. Freeman (1997) identified the following categories of ecosystem services to humankind:

1. Material inputs into economic activity (fossil fuels, minerals, animals)
2. Life-support services (breathable air, livable climate)
3. Environmental amenities used for recreation
4. Processing of waste products discharged into the environment

Available valuation methods can measure only some of these benefits: material inputs and the value of environmental amenities used for active recreation. The most important ecological effects with identifiable service flow impacts are summarized in Table 4.2.

Table 4.2
Ecological effects with identifiable and measurable human service flows

Pollutant Class	Ecosystem effect	Service flow impacted
Acidification (H_2SO_4, HNO_3)	High-elevation forest acidification resulting in dieback	Forest esthetics
	Freshwater acidification resulting in fresh water organism (e.g. fish) population decline.	Recreational Fishing
	Changes in biodiversity in terrestrial and aquatic ecosystems	Existence/Non-use values of biodiversity
Nitrogen Saturation and Eutrophication (NO_x)	Freshwater acidification resulting in fresh water organism (e.g. fish) population decline.	Recreational Fishing
	Estuarine eutrophication causing oxygen depletion and changes in nutrient cycling	Recreational and commercial fishing
	Changes in biodiversity in terrestrial and aquatic ecosystems	Existence/Non-use values of biodiversity

Source: U.S. EPA (1999)

Economic analyses of air pollution control have paid less attention to ecological benefits than direct benefits to human health. There is a complex and nonlinear relationship between ecosystem damages and air pollution, and many impacts are difficult to measure.

The most important ecological benefits of air pollution abatement include:

- Eutrophication* of estuaries associated with atmospheric nitrogen deposition
- Reduced tree growth associated with ozone exposure
- Acidification of freshwater bodies associated with atmospheric nitrogen and sulfur deposition
- Accumulation of toxics in freshwater bodies associated with atmospheric toxics deposition
- Aesthetic damages to forests associated with ozone and airborne toxics

* Eutrophication is a condition in an aquatic ecosystem where high nutrient concentrations stimulate blooms of algae. Increased eutrophication from nutrient enrichment due to human activities is one of the leading problems facing some estuaries in the Mid-Atlantic region.

Not all ecological benefits are quantifiable or can be monetized. For that reason, in valuation studies attention is often restricted to ecological impacts associated with service flows to humans, rather than broad structural changes to ecosystems. The main criteria for including service flows in valuation studies are that they must be identifiable, quantifiable and monetizable. Table 4.3 summarizes several service flows that satisfy these criteria.

Table 4.3
Candidate Endpoints for Quantitative Assessment

Ecological Effect	Endpoint	Dose-Response Functions	Economic Model
Acidification	1. Forest Aesthetics	1. Not required	1. Site-specific
	2. Recreational Fishing	2. Multiple available	2. Site-specific
	3. Existence Value of Biodiversity	3. Multiple Available	3. Site-specific
Eutrophication	1. Recreational Fishing		1. Site-specific
	2. Existence Value of Biodiversity		2. None available
Toxics Deposition	1. Forest Aesthetics	1. Multiple available	1. Site-specific
	2. Recreational Fishing	2. Multiple available	2.Site-specific
	3. Existence Value of Biodiversity	3. Multiple available	3.None available
	4. Hunting and Wildlife Aesthetics	4.Multiple Available	4. Site-specific
Multiple Pollutant Stress	1. Ecosystem aesthetics and ecosystem existence value	1. None available	1. None available

Source: U.S. EPA (1999)

There are four primary methods to monetize non-health-related benefits:

1. Hedonic property value methods
2. Travel cost methods (TCM)
3. Expressed preference methods/Contingent valuation
4. Market models

The use of hedonic property value methods and contingent valuation to monetize ecological benefits is analogous to the valuation of human health benefits. TCM exploits observed differences between travel distance and environmental quality of recreation site to estimate the monetary value of each site characteristic. Market Models study the impact of changes in ecological services on both producers and consumers of market goods that rely on these services.

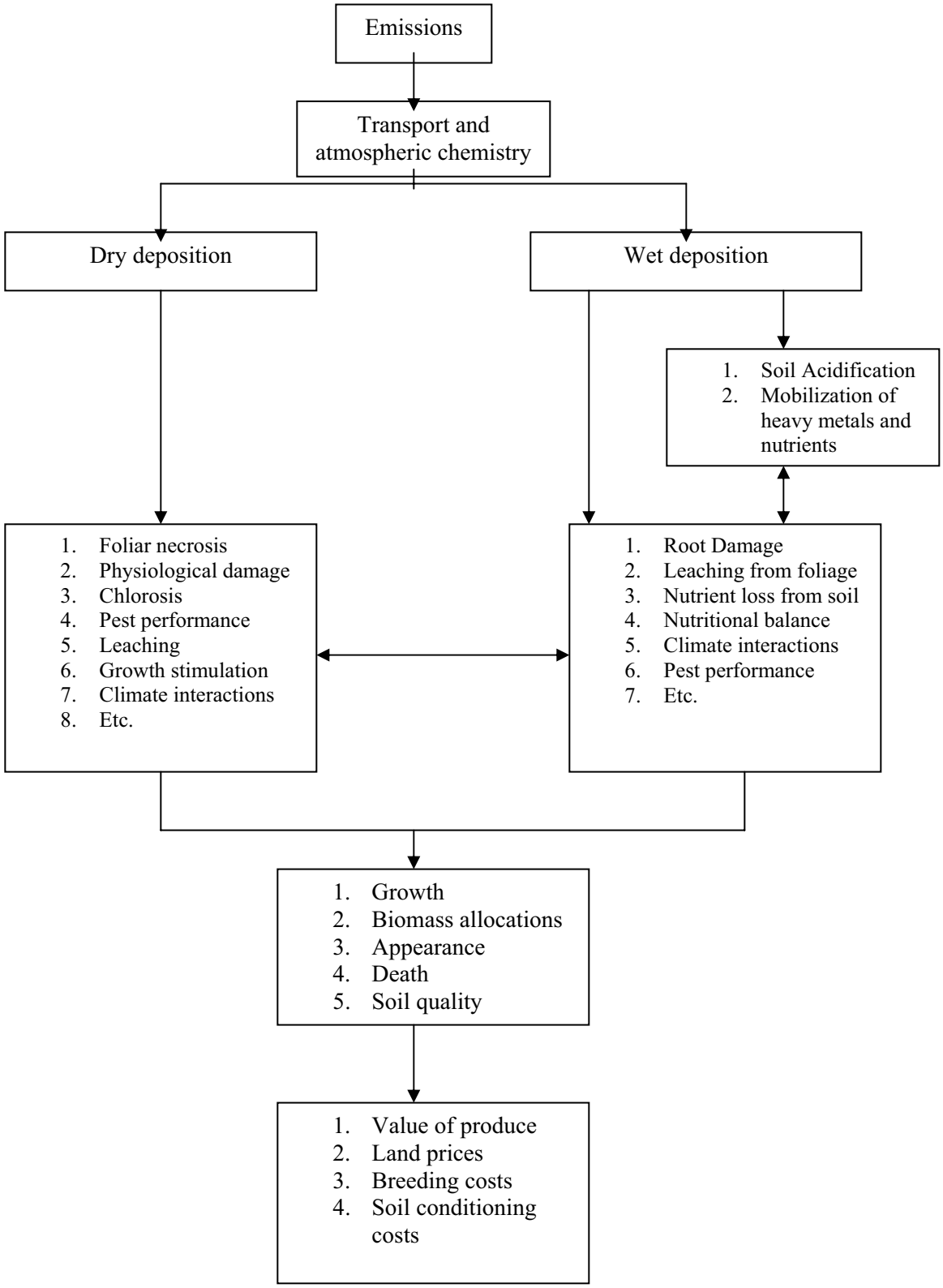
4.1. AGRICULTURAL PRODUCTIVITY BENEFITS

Air pollution has a negative impact on agricultural productivity. Research in the area has focused primarily on the economic impacts of tropospheric ozone, acidic deposition, and global climate change on commercial crops. Economic assessments of the impact of air pollution on crop losses are sometimes associated with forestry impacts. However, as Spash (1997) points out, forestry is a multi-product production system, in which the economic valuation of the impacts pertains to a much wider set of issues, including biodiversity changes, reduced aesthetics and recreation services. Some of these impacts have only non-use values, and therefore forestry damages are poorly represented in market-related production models that are commonly used to estimate agricultural productivity damages. Therefore, in what follows we review only the methodology commonly used to estimate agricultural crop losses due to air pollution.

Pollutants that have been found to have a negative impact on crop yields are ozone (O_3) and its precursor pollutants (mostly NO_x), and sulfur dioxide (SO_2). Chart 4.1 illustrates the various impact pathways of air pollution on agricultural crops. Of all the pollutants, the most extensive research in recent years has been conducted on tropospheric ozone. Ashmore (1991) concludes that although gaseous pollutants other than ozone (namely, SO_2 and NO_2) may be locally important at high concentrations, they have little economic impact on a national scale. Only minor damage to plants has been attributed to gaseous pollutants other than ozone and to sulfate and acid deposition. The National Crop Loss Assessment Network (NCLAN) program found statistically significant response to SO_2 only in soybeans and tomatoes. Herrick and Kulp (1987) report a negligible impact of SO_2 and NO_2 within the National Acid Precipitation Assessment Program (NAPAP). Ashmore (1991) finds that barley, clover, and lucerne are especially sensitive to SO_2, but these are minor crops associated with small potential economic benefits of pollution control.

Acidic deposition is an indirect impact pathway between air pollution and crop yields. While gaseous pollutants affect crops directly by foliage or above ground exposure, acidic deposition causes changes in soil chemistry due to additions of sulfur and nitrogen. The various positive (e.g. passive fertilization) and negative effects of acidic deposition could potentially neutralize each other, although the final outcome is highly dependent on edaphic conditions and crop cultivar (Spash, 1997).

Chart 4.1. Impact pathway illustrating the effects of air pollution on agricultural crops Source: Holland et al. (2002)



For example, Adams et al. (1986) included fertilization effects of nitrate deposition, and compared them with additional expense for lime used to reduce acidity. The economic analysis of a 50% increase in acidic deposition resulted in a net benefit.

Corn and soybean appear to be the most sensitive crops to acidic deposition. In addition, the available research seems to suggest that most commercial crop yields are relatively insensitive to acidic deposition on its own (Segerson, 1991). Spash (1997) concludes that crop damages from SO_2, NO_2 and acid deposition combined are 5-10% of the crop damages of ozone pollution.

Segerson (1987) has identified a number of factors that distinguish the effects of acidic deposition from those of ozone:

- Acidic deposition affects a wide range of non-market goods while ozone affects mostly commercial crops
- Acidic deposition is a dynamic pollutant that accumulates over time, while ozone is periodic. Therefore an economic analysis of ozone exposure may be based on short-term studies, while acidic deposition should be based upon assessing accumulated impacts over time.
- The impacts of acidic deposition are surrounded by a greater degree of uncertainty than those of ozone.
- Ozone pollution is a more localized problem than acidic deposition.

Ozone has been observed to cause significant damages in terms of crop yield losses at current ambient levels. Furthermore, the increased frequency and duration of hot dry weather implied by global warming will increase the concentration of tropospheric ozone from available precursors. The Table 4.1.1 below illustrates the damages to crops from ozone exposure

Table 4.1.1 Processes and characteristics of crop plants that may be affected by ozone

Growth	Development	Yield	Quality
Rate	Fruit set & development	Number	Appearance: size, shape, and color
Pattern	Branching	Mass	Storage life
	Flowering		Texture/cooking quality, Nutrient content, Viability of seeds

Source: Jacobson (1982) p. 296, Table 14.1.

Although ozone-induced quality degradation may be a significant part of total economic damages, research has almost exclusively focused on estimating changes in output resulting from air pollution. There is currently insufficient information available as to the importance of crop quality response (Spash, 1997).

The majority of economic assessments of ozone damage to crops have been at the regional level. Published studies have concentrated on two main regions of the U.S., namely, the Corn Belt (Illinois, Indiana, Iowa, Ohio and Missouri) and California.

4.1.1 QUANTIFYING AGRICULTURAL PRODUCTIVITY BENEFITS

Dose-response functions describe the relationship between ambient ozone concentrations and changes in crop yields. There are three main approaches for deriving dose-response relationships:

1. Foliar injury models
2. Secondary response data
3. Experimentation.

Foliar injury models assess visible injury symptoms, quality changes, and growth responses to air pollution, and they often require making subjective judgments by the researcher. These were the primary methods used in the early literature. Another method of dose-response function estimation is the use cross-sectional analysis of crop yield data via regression techniques. Data on outdoor pollutant concentrations, actual crop yields, and other environmental factors are need for the dose-response relationship estimation. Examples of the use of secondary response data include Leung et al. (1982) and Rowe and Chestnut (1985). Experimental approaches to study the effects of ozone on crops include the use of greenhouses, field chambers (open-top and closed-top), unenclosed plots, and the pollution gradient approach.

Using experimental methods, the National Crop Loss Assessment Network (NCLAN) developed concentration-response relationships linking ground-level ozone to leaf damage and reduced seed size in an effort to determine the effect of ozone on crop yield. Estimated minimum and maximum dose-response functions for six major crops are summarized in the table below.

Table 4.1.2 Ozone Exposure Response Functions for Selected Crops					
Ozone Index	Quantity	Crop	Dose-response Function	Median Experimental Duration (Days)	Median Duration (Months)
SUM06	Max	Cotton	$1\text{-}\exp\left(\text{-}\left(\text{index}/78\right)^{1.311}\right)$	119	4
SUM06	Min	Cotton	$1\text{-}\exp\left(\text{-}\left(\text{index}/116.8\right)^{1.523}\right)$	119	4
SUM06	Max	Field Corn	$1\text{-}\exp\left(\text{-}\left(\text{index}/92.4\right)^{2.816}\right)$	83	3
SUM06	Min	Field Corn	$1\text{-}\exp\left(\text{-}\left(\text{index}/94.2\right)^{4.307}\right)$	83	3
SUM06	Max	Grain Sorghum	$1\text{-}\exp\left(\text{-}\left(\text{index}/177.8\right)^{2.329}\right)$	85	3
SUM06	Min	Grain Sorghum	$1\text{-}\exp\left(\text{-}\left(\text{index}/177.8\right)^{2.329}\right)$	85	3
SUM06	Max	Peanut	$1\text{-}\exp\left(\text{-}\left(\text{index}/99.8\right)^{2.219}\right)$	112	4
SUM06	Min	Peanut	$1\text{-}\exp\left(\text{-}\left(\text{index}/99.8\right)^{2.219}\right)$	112	4
SUM06	Max	Soybean	$1\text{-}\exp\left(\text{-index}/131.4\right)$	104	3
SUM06	Min	Soybean	$1\text{-}\exp\left(\text{-}\left(\text{index}/299.7\right)^{1.547}\right)$	104	3
SUM06	Max	Winter Wheat	$1\text{-}\exp\left(\text{-index}/27.2\right)$	58	2
SUM06	Min	Winter Wheat	$1\text{-}\exp\left(\text{-}\left(\text{index}/72.1\right)^{2.353}\right)$	58	2
Source: USEPA (1999) Originally from Lee and Hogsett (1996)					

The commonly assumed form of the dose-response function is the Weibull function, which has the following functional form:

$$Q = \mu \cdot e^{-\left(\frac{O_3}{r}\right)^{\lambda}}$$

where Q is the observed yield, μ is the hypothetical yield at zero ozone exposure, O_3 is the ozone concentration (ppb) and γ is the ozone concentration when the yield is 0.37 μ and λ is a shape parameter. This functional form is often used in empirical analysis partly because of its biologic plausibility.

4.1.2 VALUATION OF AGRICULTURAL PRODUCTIVITY BENEFITS

The traditional approach to valuing crop losses due to air pollution was to calculate monetary equivalents of the approximated losses by multiplying decreased yields by the current market price to give a producer benefit estimate equal to total revenue. It has been shown that this method is likely to overestimate the gain to producers from ozone reductions. In addition, traditional methods often ignored farmers' reactions in terms of changing the input mix and cross-crop substitution.

In more recent empirical work agricultural production models have been used to estimate the economic costs associated with yield losses due to air pollution. These models estimate the social benefit from reduced ozone damages. The social benefit from a reduction in the ozone air pollution is the change in total surplus minus the change in deficiency/transfer payments. The social benefit, or total surplus, consists of producer surplus (the total difference between the market price and the willingness-to-supply on each unit sold) and the consumer surplus (the total difference between the market price and the willingness-to-pay on each unit bought). On one hand, a reduction in crop damage reduces costs, and hence increases the supply of the crop. This contributes to an increase in the producer surplus, ceteris paribus. On the other hand, altered levels of ozone pollution may affect the attributes of a crop, thus changing the consumers' willingness to pay, and consequently change the consumer surplus. The agricultural production model calculates the competitive equilibrium that maximizes the total surplus subject to resource constraints.

Various agricultural production models have been used in economic assessments of ozone damage. Murphy et al. (1999) use the AOM8 estimate the welfare changes due to ozone air pollution the markets for eight major crops (corn, soybeans, wheat, alfalfa hay, cotton, grain sorghum, rice, barley). The effects of a reduction in ozone concentrations are modeled as a shift in the production function—at lower ozone levels, more output is obtained from a given set of inputs. AOM8 is a modified version of the agricultural production model used in Howitt (1991). In response to output and input price changes, AOM 8 accounts for endogenous price effects and substitution of cropping activities.

Howitt et al. (1984) studied 13 crops using the California Agriculture Resources Model (CARM) to calculate consumer and producer surpluses. CARM is a quadratic programming model allowed for constrained cross-crop substitution.

The U.S. EPA (1999) study used the Agricultural Simulation Model (AGSIM©, Taylor et al., 1993). The model is able to simulate the markets (equilibrium prices and quantities) for ozone-sensitive crops. AGSIM© is an econometric-simulation model that is based on a large set of statistically estimated demand and supply equations for agricultural commodities produced in the United States.

The use of an agricultural production model requires one to specify an agricultural production function. For example, Murphy et al. (1999) use a Cobb-Douglass production

function, where land, water, nitrogen, and pesticides are the inputs. This specification assumes that a given change in ozone concentrations causes the same percentage change in output for any combination of the inputs. To estimate the changes in producer and consumer surplus, the shift in the production function is estimated based on dose-response functions for individual crops. In Murphy et al. (1999) the relationship between production levels under the baseline and the policy scenario are given by the following formula:

$$Q_i^P = (1 + \frac{QGAIN\%_i}{100})Q_i^B$$

Where Q_i^S and Q_i^B are production levels of crop i under the policy scenario and the baseline scenario, respectively, and $QGAIN\%_i$ is the percentage change in the yield of crop it resulting from a reductions in ambient ozone concentrations induced by the policy. The percentage change in the output, $QGAIN\%_i$, is estimated using dose-response functions. Each ozone reduction scenario results in a unique set of optimal input quantities, equilibrium output prices and quantities, and welfare measures (total, consumer, and producer surplus).

4.2 RECREATIONAL AND COMMERCIAL FISHING BENEFITS

Recreational and commercial fishing benefits form a subgroup of ecological benefits of air pollution. The theoretical basis for valuing ecological benefits in general, and recreational and commercial fishing benefits in particular, is that the natural environment provides us with services that we value (Freeman, 1997). There are no suitable methods to comprehensively value many of these service flows (e.g. breathable air, livable climate). Therefore in valuation studies, we are limited to valuing services flows that are either material inputs into our economy, or provide amenities associated with marketed services (e.g. recreation).

Three types of pollution are associated with commercial and recreational fishing: acidification, nitrogen eutrophication, and toxics deposition. Acidification, or acid deposition, is probably the best-studied effect of air pollution on ecosystems. The main cause of acidification is acidic precipitation in the form of sulfuric acid (H_2SO_4) and nitric acid (HNO_3). These acids are formed from sulfur dioxide (SO_2) and nitrogen oxides (NO_x) found in the atmosphere. Electric power plants are among the primary point sources of SO_2. On the other hand, a large share of NO_x is from non-point sources (transportation), and therefore anthropogenic NO_x is more dispersed in the atmosphere compared with anthropogenic SO_2. Deposition occurs via three main pathways: (1) wet deposition, where the pollutant is dissolved in precipitation, (2) dry deposition, which is a direct form of deposition of gases and particles to any surface, and (3) cloud-water deposition, when cloud or water droplets are intercepted by vegetation. Since most of the precipitation falls on the terrestrial part of the catchments, soil properties are generally

strongly associated with water quality. Consequently, acidification resulting from acid deposition usually occurs in areas with acidic soils. Throughout the world freshwater acidification is the most serious in eastern parts of the United States and Canada, and in Europe, particularly in Scandinavia. Reductions of anthropogenic SO_2 and NO_x emissions in Europe in recent years have resulted in an improvement in acidified water bodies, however, the same trend has not been observed in the United States (Stoddard et al., 1999). It is believed that it may take ecosystems several decades to recover from the impact of acidification even after emissions have been cut.

Eutrophication is the result nitrogen deposition leading to excessive nitrogen enrichment of aquatic ecosystems, and it may adversely affect the biogeochemical cycles of watersheds. Atmospheric nitrogen is deposited into water bodies through dry and wet deposition. Excessive eutrophication can lead, for example, to massive algae booms, which in turn reduces the oxygen levels and leads to habitat loss. It is estimates that 86% of the East Coast Estuaries are susceptible to eutrophication (U.S. EPA, 1997).

Toxics deposition involves hazardous air pollutants (HAPs), as defined by the Clean Air Act: mercury, polychlorinated biphenyls (PCBs), chlordane, dioxins, and dichlorodiphenyltrichloro-ethane (DDT). When considering air pollution from power plants, the most important of these pollutants is mercury. Much of mercury found in ecosystems comes from natural sources. Mercury accumulates in fish, birds, and mammals, and it may be dangerous to humans when the concentration exceeds a critical level. Mercury-based statewide fish consumption advisories are fairly common in the United States.

Table 4.2.1 below summarizes the main recreational and commercial fishing impacts associated with acidification, eutrophication, and toxics deposition.

Table 4.2.1 Recreational and commercial fishing can be associated with the following ecological impacts of air pollution:

Pollutant Class	Ecosystem Effect	Service Flow
Acidification (H_2SO_4, HNO_3)	Freshwater acidifcation resulting in fish (and other aquatic organism) decline	Recreational Fishing
Nitrogen Eutrophication (NO_x)	Freshwater acidifcation resulting in fish (and other aquatic organism) decline	Recreational Fishing
Toxics Deposition (Mercury, Dioxin)	Aquatic bioaccumulation of mercury and dioxin	Recreational and Commercial Fishing

Source: U.S. EPA (1999)

4.2.1 Quantifying Recreational and Commercial Fishing Benefits

Following emissions modeling, and transport and deposition modeling, the next step in the process of quantifying the benefits to recreational fisheries is the use of an exposure

model. Unfortunately, comprehensive models to quantify the impacts of acidification, eutrophication, and toxics deposition are currently not available.

To quantify the impact of acid deposition on fisheries, US EPA (1999) uses a region-specific model to quantify the effects of acidification on freshwater fish populations: Model of Acidification of Groundwater Catchments – MAGIC (Cosby et. al. (1985a,b). MAGIC is calibrated to the watershed of an individual lake or stream and then used to simulate the response of that system to changes in atmospheric deposition. The model simulates the effects of acid deposition on both soils and surface waters. The simulation typically involves seasonal or annual time steps and is implemented on decadal or centennial time scales.

Quantifying Eutrophication

When atmospheric nitrogen is deposited in estuaries, it can lead to eutrophication. Estimation of a dose-response relationship between nitrogen loading and water quality changes is complicated because of the dynamic nature of ecosystems. Most likely, these dose-response functions are nonlinear with a threshold. Unfortunately, universally transferable dose-response functions for quantifying the effects of eutrophication have not yet been developed. USEPA (1999) study quantified deposition-related nitrogen loadings for three estuaries (Chesapeake Bay, Long Island Sound, Tampa Bay) using GIS-related methods. Data on nitrogen deposition, together with information on abatement options to reduce excess nutrient loads, was used for valuation purposes. In addition, USEPA (1999) used specific biophysical indicators of estuarine health to quantify the benefits. This approach is useful when there is a direct link between the biophysical indicator and the ecological service flows. USEPA (1999) used the properties of the seagrass bed, which provides habitat for variety organisms, and have been shown to decline with increased nitrogen deposition, as an indicator.

Quantifying Toxics Deposition

Most damages to ecosystems are caused by five hazardous air pollutants (HAP): mercury, PCBs, dioxins, DDT, and chlordane. The mechanism of ecosystem responses to toxic contamination is poorly understood. Furthermore, service flow impacts of ecosystem damages are difficult to observe because HAPs persist in aquatic ecosystems for a long time. A comprehensive quantitative analysis with the available models and data is currently not possible.

4.2.2 Valuation of Recreational and Commercial Fishing Benefits

Unlike commercial fishing, recreational fishing is to a large part a non-market activity. Although most states charge a license fee for recreational fishing, the license fee itself is believed not to reflect the true willingness-to-pay (WTP) for recreational fishing. Marginal WTP for recreational fishing is a function of catch attributes (e.g. number and the average size of fish caught) and other determinants. Environmental factors indirectly affect WTP for recreational fishing by affecting the catch attributes. The total value of recreational fishing to the angler can be measured by the consumer surplus, which is the difference between WTP and the actual amount they pay or the cost they incur for the recreational fishing day. Consumer surplus is measured by the area below the demand curve and above the price or the cost of a recreational fishing day.

There is an extensive literature on valuation of fishing opportunities by anglers. In the valuation literature, two primary methods have been used most often to deduce the value of recreational fishing: travel cost method (TCM), and contingent valuation method (CVM). TCM uses observed travel costs to visit a fishing site and per-capita visitation rates to deduce the demand for recreational fishing. On the other hand, CVM is questionnaire-based method, where anglers are asked hypothetical question about how much they would be willing to pay for a day of fishing.

TCM cannot be used to measure willingness-to-accept (WTA) some degradation in an environmental amenity (i.e. compensation demanded for an environmental damage). Hence, TCM cannot be used to estimate the value of loss of fishing opportunity due to air pollution. Furthermore, the use of CVM in general has generated controversy in the valuation literature. CVM is subject to an inherent bias due to its hypothetical nature. Study participants are often subjected to an unfamiliar market context, or they may not be fully aware of the characteristics of the good in question, or their own budget constraints. Some critics of CVM have pointed out that estimates of WTP obtained using CVM may not reflect the true WTP for the non-market good, but they rather reflect the WTP pay for moral satisfaction. The answers from CVM studies may be biased because of passive-use motives, such as the “warm glow” effect (Andreoni, 1989). Individuals’ responses to WTP questions serve the same function as charitable contributions, and people are assumed to get a "warm glow" from giving. Some economists do not fully recognize “warm glow” as an economic value.

In contrast to many earlier studies utilizing TCM or CVM, Snyder et al. (2003) use a revealed-preference approach to estimate the value of recreational fishing. Their method to estimate WTP for a recreational fishing day is based on observed fishing license sales. Unlike CVM and TCM that require extensive micro-level data, Snyder et al. (2003) are able to use aggregate, state-level data for their estimation. The following simple model describes the methodology used in Snyder et al (2003). A representative consumer’s utility depends on the number of recreational fishing days, X, the fishing license L, and all other goods denoted by a composite good Z.

$$U = U(X, L, Z)$$

Because X and L are complementary, we can write the following:

$$\frac{\partial U(X, 0, Z)}{\partial X} = 0$$

$$\frac{\partial U(0, L, Z)}{\partial X} = 0$$

That is, the marginal utility of a fishing license or a recreational fishing day alone is zero. The demand for annual fishing licenses can be estimated from the observable data on license sales. By measuring the appropriate area under the demand curve, one can estimate the average benefits per license, and consequently, the value of a recreational fishing day.

Several problems may arise when using this method. One is that the assumed complementarity between the fishing license and recreational fishing day holds only in the absence of illegal fishing. A comprehensive economic model of consumer behavior would account for illegal fishing by including the "price" of illegal fishing in the estimation process. As Snyder et al. (2003) note, in most cases, fines are set by courts, and therefore omitting fines should not bias the analysis, as there is no apparent correlation between license fees and fines. Another potential problem associated with the method of Snyder et al. (2003) is price endogeneity, that is, that causality runs not only from price to quantity, but vice versa. To address possible price endogeneity, the authors use the instrumental variable (IV) estimation technique, using instruments that are exogenous to the demand for fishing licenses. The set of instruments includes variables that are indicative bureaucratic and political proclivities of states, such as the size of the government, and the degree or regulation and taxation.

Snyder et al. (2003) obtain estimates of the value of a recreational fishing day for 48 U.S. states. Table 4.2.2 summarizes the results for Mid-Atlantic states that are most likely to be affected by the New Jersey RPS.

Table 4.2.2 Estimates of the value of a freshwater recreational fishing day for selected states Snyder et al. (2003) (2000$'s)

State	Linear IV	Semi-log IV	Log-log IV cutoff $32.8	Log-log IV cutoff $100	Log-log IV cutoff $200	Log-log IV cutoff $500	90% confidence interval based on semi-log		90% confidence interval based on log-log ($200 cutoff)	
New Jersey	1.33	1.29	0.23	0.37	0.50	0.74	0.25	6.65	0.00	3.21
New York	1.45	1.29	0.32	0.51	0.69	1.03	0.30	5.59	0.01	3.49
Pennsylvania	2.21	3.00	2.01	3.26	4.40	6.56	0.57	15.87	2.06	7.43
Maryland	1.66	1.38	0.34	0.55	0.74	1.11	0.35	5.44	0.01	3.63
Delaware	1.08	0.79	0.18	0.30	0.40	0.60	0.23	2.74	0.00	2.13

Notes:

i. Estimates are based on instrumental variables regressions on data from 1975-1989

ii. Models of demand for three functional forms are estimated: linear, multiplicative (log-log) and semi-log. For each functional form, two specifications are reported: one includes all relevant substitutes of resident annual licenses, and the other includes only the price of short-term Type 1 licenses and dummy variables for each year.

iii. Cutoff values are used as upper limits to integrate the demand function.

A comparison of Snyder et al. (2003) estimates with the values from other studies reveals that there is a considerable geographic variation in the estimated value of recreational fishing. Moreover, the estimates are significantly lower than those reported in other studies employing TCM or CVM. The differences could be due to methodological differences, as well as, to the elimination of the biases in TCM and CVM.

Table 4.2.3 Comparison of Snyder et al. (2003) estimates with other studies					
Study	Estimation Method	Study Location	Type of Fishing	Valuation (2000 $'s)	Valuation Snyder et al. (2003)[i] (2000 $'s)
King and Hof (1985)	TCM	Alabama	Trout	24.57	5.86 (5.22,32.14)
Miller and Hay (1980)	TCM	Arizona	All	73.14	2.90 (1.09, 5.14)
Walsh et. al. (1980)	CVM	Colorado	Cold water	22.01	10.40 (10.01, 30.38
Ziemer et.al. (1980)	TCM	Georgia	Warm water	27.65	2.92 (2.80, 5.59)
Miller and Hay (1980)	TCM	Idaho	All	56.42	11.12 (10.80, 25.18)
Loomis and Sorg (1986)	TCM	Idaho	Cold water Warm water	45.59 47.04	11.12 (10.80, 25.18)
Miller and Hay (1980)	TCM	Maine	All	48.06	4.43 (2.73, 6.88)
Miller and Hay (1980)	TCM	Minnesota	All	60.60	18.80 (13.34, 305.88)
Haas & Weithman (1982)	TCM	Missouri	Trout	27.97	4.86 (4.62, 8.06)
Dutta (1984)	TCM	Ohio	Cold water	8.73	3.51 (2.60, 5.24)
Brown and Shalloof (1984)	TCM	Oregon	Salmon Steelhead	36.47 49.72	12.97 (12.29, 26.94)
Kealy and Bishop (1986)	TCM	Wisconsin	All	51.60	11.55 (10.37, 52.00)

Notes:
i. Snyder et al. (2003) estimates are based on the log-log instrumental variable specification with $200 cutoff; 90% confidence interval in brackets

Another limitation of many early studies is that they do not include a direct measure of water quality. A notable exception is Montgomery and Needelman (1997) that consider the special case of toxic contamination of fisheries. Toxic contamination is a special case of pollution, because contaminants in fish become dangerous to humans eating fish before they result in a decline in fish population. In addition, through health advisories the public is better informed about incidences of toxic contamination than other forms of pollution (e.g. acidification or eutrophication).

Montgomery and Needelman (1997) employ the Random Utility Model, which is a site-choice model. A brief description of the RUM model follows. Utility associated with recreational fishing for individual i in fishing site j is given by the following equation:

$$U_{ij}^F = V_{ij}^F + \varepsilon_{ij}^F$$

where V_{ij}^F is the observable portion of the utility function, and ε_{ij}^F is a random error. Standard RUM models assume that V_{ij}^F is a linear function of income, M_i, cost of visiting the site, P_{ij}, and a vector of site characteristics, X_j.

$$V_{ij}^F = \beta_M (M_i - P_{ij}) + \beta_X X_j$$

where β_M and β_X are parameters to be estimated. Using the parameter estimates we can estimate the inclusive value, I_i, which is the maximum utility that an individual taking a trip receives.

$$I_i = \ln \sum_{j=1}^{J} e^{\mu\beta_X X_{ij} - \mu\beta_M P_{ij}}$$

The utility of not fishing is represented by the following equations.

$$U_i^N = V_i^N + \varepsilon_i^N$$

$$V_i^N = \beta_M M_i + \beta_Z Z_i$$

Given the set of individual attributes, Z_i, the probability that an individual will go fishing on a given day is given by the following formula:

$$\Pr_i(fish = 1) = \frac{e^{\frac{1}{\mu} I_i}}{e^{\frac{1}{\mu} I_i} + e^{\beta_Z Z_i}}$$

The economic value for an individual of improved water quality (compensating variation) can be calculated as follows:

$$4\,CV_{it} = \frac{\ln(e^{\frac{1}{\mu}\hat{I}_i^2} + e^{\hat{\beta}_Z Z_i}) - \ln(e^{\frac{1}{\mu}\hat{I}_i^1} + e^{\hat{\beta}_Z Z_i})}{\hat{\beta}_M}$$

Table 4.2.3 Valuation studies of recreational fishing using RUM			
Study	Location	Study Period	Type of Fishing
Morey et al. (2001)	Maine rivers	1988	Salmon fishing
Ahn et al. (2000)	North Carolina mountain streams	1996	Trout fishing
Jakus et al. (1998)	Tennessee reservoirs	1997	Recreational fishing
Lupi and Hoehn (1998)	Great Lakes, Michigan	1994	Trout and salmon fishing
Parsons et al. (1998)	Maine lakes and rivers	1989	Recreational fishing
Pendleton and Mendelsohn (1998)	New England lakes	1989	Recreational fishing
Schumann (1998)	North Carolina	1987-1990	Ocean Fishing
Train (1998)	Montana rivers and lakes	1987-1990	Recreational fishing
Greene et al. (1997)	Tampa Bay, Florida	1991-1992	Recreational fishing
Hoehn et al. (1997)	Michigan lakes and rivers	1994-1995	Recreational fishing
Montgomery and Needelman (1997)	New York lakes	1989	Recreational Fishing
Feather et al. (1995)	Minnesota lakes	1989	Recreational fishing
McConnell and Strand (1994)	Mid- to South-Atlantic	1987-1988	Recreational ocean fishing

4.3 BIODIVERSITY BENEFITS

Biodiversity is a valuable environmental amenity. A number of human actions, including anthropogenic air pollution, have led to a dramatic decline in biodiversity across the globe (Pimm et al., 2001). Biodiversity refers not just to an accumulation of species in a given area, but it also incorporates the ecological and evolutionary interactions among them (Armsworth et al., 2004).

The first step in estimating biodiversity benefits is defining biodiversity. Biodiversity encompasses four levels as it is summarized in the table below:

Table 4.3.1

Type of Biodiversity	Physical Expression
Genetic	Genes, nucleotides, chromosomes, individuals
Species	Kingdom, phyla, families, genera, subspecies, populations
Ecosystem	Bioregions, landscapes, habitats
Functional	Ecosystem, functional, robustness ecosystem resilience services goods

Source: Turner et al. (1999)

Genetic biodiversity is the most basic level, and it refers to the information represented in the DNA of living organisms. Species-level biodiversity refers to the variety of species in a given area. Because only a small fraction of the estimated 5-30 million species currently living on the earth (Wilson, 1988) have been identified and described, empirical estimates of the species-level biodiversity are often surrounded by a great degree of uncertainty. Community-level biodiversity is important, because it is believed that species-level diversity enhances the productivity and stability of ecosystems (Nunes and van den Bergh., 2001, Odum, 1950). However, recent studies suggest that no pattern or determinate relationship may exist between species-level diversity and stability of ecosystems (Nunes and van den Bergh. 2001, Johnson et al. 1996). Functional diversity, or the ecosystem's functional robustness, refers to the ability of the ecosystem to absorb external shocks. Unfortunately, the ecosystem's functional diversity is still poorly understood (Nunes and van den Bergh, 2001).

Human threats to biodiversity include activities causing habitat loss (conversion, degradation or fragmentation) and climate change, harvesting, as well as the introduction of exotic species that by becoming dominant competitors or effective predators may drive many native species to extinction.

The electric utility sector contributes to habitat degradation (acidic deposition and eutrophication) by emitting nitrogen oxides (NO_x) and sulfur oxides (SO_x), as well as to climate change by emitting greenhouse gases into the atmosphere.

Empirical estimates of Morse et al. (1995) and Field et al. (1999) of the impact of climate change on biodiversity illustrate the magnitude of threats to biodiversity: 4 °F average temperature increase can reduce the number of all species in California by 5%-10%.

4.3.1 Quantifying Biodiversity Benefits

The following discussion on quantifying biodiversity benefits is based on Armsworth et al. (2004). The traditional approach to measuring biodiversity has focuses on species-level biodiversity, which can be measured in two ways:

- *Richness* – number of species in a given area
- *Evenness* – how well distributed abundance or biomass is among species within a community

Evenness is the greatest when species are equally abundant. For example an area that has a total population of 100 of 10 different species, each comprising of 10 individuals, is more diverse than a community of 1 species with 91 individuals and 9 other species with one individual each.

A diversity index is an overall measure of diversity that usually combines aspects of richness and evenness. One of the most commonly used diversity index is the Shannon-Weiner index (H') defined below.

$$H' = -\sum_{i=1}^{S} p_i \ln(p_i)$$

where the summation is over all species (i.e., S is the total number of species at site), and p_i is the relative abundance of species it (i.e., p_i is the number of individuals of species divided by the total number of individuals of all species at the site). H' is high if there are many species, or if evenness is high.

Example Calculation of H':
Suppose we study a 1-acre area in a forest and have counted 240 redbud trees, 120 post oak trees, 40 black hickory tree, and 320 red oak trees. Species richness calculations are summarized in the table below:

Species (i)	N_i	p_i	$ln(p_i)$	$p_i \ln(p_i)$
Redbud trees (1)	240	0.333	-1.100	-0.366
Post oak tree (2)	120	0.167	-1.792	-0.298
Black hickory tree (3)	40	0.056	-2.882	-0.161
Red oak tree (4)	320	0.444	-0.811	-0.361
Total	720	1.000		-1.186

Estimating relative abundances for all species can be time-consuming and difficult, therefore species-level richness is often used as a proxy for species-level biodiversity. In contrast to the Shannon-Weiner index this simplification places relatively large weight on rare species. Typically, measures of species-level diversity are not applied to all species at site, but rather to a particular taxonomic group, such as, mammals, insects, or plants (Armsworth et al. 2004)

The choice of the spatial scale is also important, because richness increases with the size of the area. The appropriate scale is typically an economically meaningful scale (e.g., individual land parcel) or an ecologically meaningful scale (e.g., habitat zone).

Once the spatial scale has been chosen, there are three aspects of biodiversity to consider (Whittaker 1972, Schluter and Ricklefs, 1993):

- *α-diversity* – the "local" diversity within each site
- β-diversity – the change in species composition from one site to another
- γ-diversity – the "total" diversity measured over the entire suite of sites being considered

When ecological data are not available, ecological or biological production functions may be used to approximate the changes to biodiversity. One of the most robust and useful ecological patterns that researchers have observed is the species-area relationship. This relationship is often approximated by a power-law formula:

$$S = cA^z$$

where c and z are positive constants.

This relationship describes a static pattern of biodiversity, and it is not informative about the composition of the local community. The simplest representation of the dynamics of a closed community takes the form:

$\dot{N}_i = N_i f^i(N_1, \ldots, N_S)$
for $i = 1, \ldots, S$

S = number of interacting species, f^i = per capita growth rate of each species; f^i can depend on all species' densities. The relevant partial densities indicate whether the interaction between any two species in the community is cooperative ($f^i_{N_j} > 0, f^j_{N_i} > 0$), predatory or parasitic ($f^i_{N_j} < 0, f^j_{N_i} > 0$) or competitive ($f^i_{N_j} < 0, f^j_{N_i} > 0$). A few functional forms of ecological production functions have been reported in the literature (e.g. Roughgarden, 1997).

4.3.2 Valuation of Biodiversity Benefits

The monetization of biodiversity benefits requires one to assess what it is about biodiversity that consumers value. In general, consumers' benefit can be divided into *use value* (direct such as tourism or indirect such as pollination) or *non-use* (intrinsic or existence) *value*. Direct benefits to consumers arise in two important ways:

- *Service flows* – Ecosystems provide valuable services to society, such as water purification in natural watersheds, prevention of soil erosion and carbon sequestration by standing forests, and recreational services such as ecotourism and birdwatching. The service flow approach to valuation dictates that investments in preserving or restoring biodiversity need to deliver a competitive return relative to other investment opportunities within the economy, and hence it does not necessarily maximize α, β or γ diversity (Armsworth, 2004).\

- *Bet hedging* – Conserving biodiversity provides society with a bet-hedge against unforeseen circumstances. For example if society were overly reliant on monocultured ecosystems, it would be vulnerable to catastrophic losses in service provision due to disease outbreaks. Hence there is a bet-hedging benefit to conserving γ diversity.

Nonuse or *existence value* of biodiversity refers to the utility consumers derive from knowing that certain species (still) exist.

Nunes and van den Bergh (2001) critically evaluate a number of biodiversity valuation studies at each level of biodiversity value contained in Table 4.3.1. The authors conclude that available economic valuation estimates should be regarded as providing a very

incomplete perspective on the value of biodiversity changes, and they provide at best the lower bounds on that value. The tables below summarize the results form the valuation studies that have been performed in North America. Table 4.3.2 lists the results from contingent valuation studies estimating the individual WTP to avoid the loss of a particular species. The main shortcoming of these single-species valuation studies is that they do not account for species substitution and complementary effects.

Table 4.3.2 Biodiversity value estimates from single-species valuation studies

Author(s)	Study	Mean WTP estimates (per household per year)
Stevens et al. (1997)	Restoration of the Atlantic salmon in one river, Massachussetts	$14.38-21.40
Loomis and Larson (1994)	Conservation of the Gray Whale, US	$16–18
Loomis and Helfand (1993)	Conservation of various single species, US	From $13 for the Sea Turtle to $25 for the Bald Eagle
Van Kooten (1993)	Conservation of waterfowl habitat in Canada's wetlands region	$50–60 (per acre)
Bower and Stoll (1988)	Conservation of the Whooping Crane	$21–141
Boyle and Bishop (1987)	Two endangered species in Wisconsin: the Bald Eagle and the Striped Shiner	From $5 for the Striped Shiner to $28 for the Bald Eagle
Brookshire et al. (1983)	Grizzly Bear and Bighorn Sheep in Wyoming	From $10 for the Grizzly Bear to $16 for the Bighorn Sheep

Source: Nunes and van den Bergh (2001)

Multiple-species valuation studies account for all related species, and the resulting estimates are in general higher than those of single-species studies. Multiple-species valuation studies are summarized in Table 4.3.3 below.

Table 4.3.3 Biodiversity value estimates from multiple-species valuation studies		
Author(s)	Study	Mean WTP Estimates (per household per year)
Desvousges et al. (1993)	Conservation of the migratory Waterfowl in the Central Flyway	$59–71
Whitehead (1993)	Conservation program for coastal nongame wildlife	$15
Duffield and Patterson (1992)	Conservation of fisheries in Montana Rivers	$2–4 (for residents) $12–17 (for non residents)
Halstead et al. (1992)	Preservation of the Bald Eagle, Coyote and Wild Turkey in New England	$15
Samples and Hollyer (1989)	Preservation of the Monk Seal and Humpback Whale	$9.6–13.8
Hageman (1985)	Preservation of threatened and endangered species populations in the US	$17.73–23.95
Source: Nunes and van den Bergh (2001)		

Table 4.3.4 summarizes estimates from valuation studies that link the value of biodiversity to the value of natural habitat conservation.

Table 4.3.4 Biodiversity value estimates from natural habitat valuation studies		
Author(s)	Study	Mean WTP estimates (per household)
Richer (1995)	Desert protection in California, US	$101
Kealy and Turner (1993)	Preservation of the aquatic system in the Adirondack Region, US	$12–18

Table 4.3.4 Biodiversity value estimates from natural habitat valuation studies (continued)		
Author(s)	Study	Mean WTP estimates (per household)
Hoehn and Loomis (1993)	Enhancing wetlands and habitat in San Joaquin valley in California, US	$96–184 (single program)
Diamond et al. (1993)	Protection of wilderness areas in Colorado, Idaho, Montana, and Wyoming, US	$29–66
Silberman et al. (1992)	Protection of beach ecosystems, New Jersey, US	$9.26–15.1
Boyle (1990)	Preservation of the Illinois Beach State Nature Reserve, US	$37–41
Loomis (1989)	Preservation of the Mono Lake, California, US	$4–11
Smith and Desvousges (1986)	Preservation of water quality in the Monongahela River Basin, US	$21–58 (for users) $14–53 (for nonusers)
Mitchell and Carson (1984)	Preservation of water quality for all rivers and lakes, US	$242
Walsh et al. (1984)	Protection of wilderness areas in Colorado, US	$32
Source: Nunes and van den Bergh (2001)		

Table 4.3.5 summarizes estimates from valuation studies that link the value of biodiversity to the value of natural areas with high tourism and outdoor recreation demand.

Table 4.3.5 Biodiversity value estimates from ecosystem functions and services valuation studies			
Author(s)	Study	Measurement method	Estimates
Laughland et al. (1996)	Value of a water supply in Milesburg, Pennsylvania, US	Averting behavior	$14 and 36 per household

Table 4.3.5 Biodiversity value estimates from ecosystem functions and services valuation studies (continued)			
Author(s)	Study	Measurement method	Estimates
Abdalla et al. (1992)	Groundwater ecosystem in Perkasie, Pennsylvania, US	Averting behavior	$61 313–131 334
McClelland et al. (1992)	Protection of groundwater program, US	Contingent valuation	$7–22
Torell et al. (1990)	Water in-storage on the high plains aquifer, US	Production function	$9.5–1.09 per acre-foot
Ribaudo (1989a,b)	Water quality benefits in ten regions in US	Averting behavior	$4.4 billion
Huszar (1989)	Value of wind erosion costs to households in New Mexico, US	Replacement costs	$454 million per year
Holmes (1988)	Value of the impact of water turbidity due to soil erosion on the water treatment, US	Replacement costs	$35–661 million annually
Walker and Young (1986)	Value of soil erosion on (loss) agriculture revenue in the Palouse region, US	Production function	$4 and 6 per acre
Source: Nunes and van den Bergh (2001)			

Finally, Table 4.3.6 provides ranges of estimates for the various levels of biodiversity value derived from the studies reviewed by Nunes and van den Bergh (2001).

Table 4.3.6 Summary of biodiversity values by biodiversity level			
Biodiversity level	Biodiversity value type	Value ranges	Method(s) selected
Genetic and species diversity	Bioprospecting	From $175 000 to $3.2 million	Market contracts
	Single species	From $5 to 126	Contingent valuation
	Multiple species	From $18 to 194	Contingent valuation
Ecosystems and natural habitat diversity	Terrestrial habitat (non-use value)	From $27 to 101	Contingent valuation
	Coastal habitat (non-use value)	From $9 to 51	Contingent valuation
	Wetland habitat (non-use value)	From $8 to 96	Contingent valuation
	Natural areas habitat (recreation)	From $23 per trip to 23 million per year	Travel cost, tourism revenues
Ecosystems and functional diversity	Wetland life-support	From $0.4 to 1.2 million	Replacement costs
	Soil and wind erosion protection	Up to $454 million per year	Replacement costs, hedonic price, production function
	Water quality	From $35 to 661 million per year	Replacement costs, averting expenditure
Source: Nunes and van den Bergh (2001)			

4.4 FORESTRY BENEFITS

Air pollution has been recognized as a potential problem for forests for a long time. Sulfur dioxide, fluorides, heavy metals and ozone pose the greatest threat to forest ecosystems. In the past, sulfur emissions, that cause acid rain, were the primary concern, but in recent decades massive efforts to reduce this pollutant have been largely successful. Today, in terms forestry impacts ozone may be the pollutant associated with the greatest potential benefits.

Scientific evidence suggests that elevated tropospheric ozone levels disrupt vegetation growth, and interfere with the respiratory function of plants carried out by photosynthesis even at concentrations below current air quality standards (Wang et al., 1986; Reich and

Amudson, 1985). Sometimes ozone injury to plants has observable effects such as yellowing or stippling of leaves, but negative impacts of ozone often occur without accompanying visible symptoms.

Ambient ozone enters the plant through pores in the leaf or needle called stomata, where most of the plant's metabolic and respiratory activity occurs. Once ozone enters these stomata, it initiates a chain reaction that destroys or damages plant proteins and enzymes, as well as the fatty chemicals that help form cell membranes. Plants continue to suffer damage long after the ozone exposure episode is over. Furthermore some researchers have suggested that there are synergies between ozone and acid deposition (Hewitt, 1990).

Ozone damage to forests is a common problem in many parts of the eastern U.S. Particularly sensitive species to ozone are the poplars (Populus spp.), white pine (Pinus strobus), and the oak family (Quercus spp.).

Another serious threat to forest ecosystems is acid deposition in the form of nitrogen acids due to nitrogen oxides emissions. Aluminum is naturally present in forest soils in the form of chemical compounds that are harmless to living organisms. Nitrogen acids cause ions of aluminum to become mobile in soil, and its toxic form, aluminum is taken up into the tree's roots. This may result in reduced root growth, which reduces the tree's ability to take up water and withstand drought. Excess nitrogen is also absorbed directly from the air through the leaves during fog and low clouds. If ozone is present in sufficient concentrations, exposure to this oxidant can damage the leaves, damaging respiration processes of the organism.

Given the evidence on damage to forests and the size of the forest cover, forestry benefits seem to play an important role in total benefits due to reduced air pollution in the northeastern United States. According to Mid-Atlantic Integrated Assessment (MAIA) – multi-agency effort headed by the USEPA to assess the health and sustainability of ecosystems – forests cover 61% of the total land area in the MAIA region[9]. Ninety-five percent of the region's forests are classified as timberland. The vast majority (79%) of timberlands is owned by nonindustrial private landowners, while the forest industry owns approximately 7%. Hardwood forests dominate the MAIA region. For the region as a whole, oak /hickory is the predominant forest type. Other dominant forest types in the region are northern hardwoods, loblolly/shortleaf pine, and oak/pine.

4.4.1 Quantifying Forestry Benefits

Due to the different life cycles involved, the assessment of forest damage is substantially more difficult than that of agricultural crop damage. On one hand trees live for a long time, which makes the study of pollution impacts much more difficult. On the other hand,

[9] The MAIA study region includes Delaware, Maryland, Pennsylvania, Virginia, and West Virginia, and parts of New Jersey, New York, and North Carolina.

unlike agricultural soils, which are effectively managed through annual cycles, forest soils are much less disturbed which leads to an accumulation of acidification impacts.

USEPA (1999) uses the PnET-II model to estimate the impacts of troposhperic ozone on commercial timber growth. The PnET model simulates the cycles of carbon, water, and nitrogen through forest ecosystems. Model inputs of monthly weather data and nitrogen inputs are used to predict photosynthesis, evapotranspiration, and nitrogen cycling on a monthly time-step for several forest types.

4.4.2 Valuation of Forestry Benefits

The valuation techniques of forestry benefits can be grouped into three categories:

1. direct market prices
2. indirect market prices
3. hypothetical values

Methods using direct market prices are based on actual prices, and consequently they do not reflect some benefits (e.g. preservation of biodiversity) that the market participants did not take into account in their decision-making. Methods utilizing indirect market prices include hedonic property values, the travel costs, opportunity costs, surrogate prices and replacement costs (Cavatassi, 2004). The opportunity cost method uses the market price of the best alternative forgone to provide a lower bound on forestry benefits. Surrogate prices methods use the market price of a close substitute as a proxy for the benefits. A surrogate market approach is used by methods using hypothetical values. Two methods that belong to this category are the contingent valuation method, and conjoint analysis.

As described in the pervious sections, contingent valuation method uses surveys that ask hypothetical questions to estimate economic values. Conjoint analysis estimates values by asking people by asking people questions across a range of features or attributes of a forest[ϕ]. Forestry benefits may be grouped into three categories for valuation purposes: on-site private benefits, on-site public benefits, and global benefits. On-site private benefits include timber productions, agricultural and other agroforestry products, and non-timber forest products (e.g. mushrooms, medicinal plants, honey, fruits, nuts, etc.), and recreation and tourism. On-site public benefits include watershed protection, agricultural productivity enhancement, nutrient cycling, microclimate regulation, and aesthetic, cultural, and spiritual values. Global benefits include carbon sequestration, and biodiversity conservation.

Values derived for some of these benefits are not transferable, and therefore most valuation studies restrict attention to on-site benefits such as timber production. These benefits are usually estimated using market models. For example, USEPA (1999) used

[ϕ] Cavatassi (2004)

the USDA Forest Service Timber Assessment Market Model to estimate market changes that result from reduced timber growth.

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5. INDIRECT BENEFITS TO HUMANS THROUGH NONLIVING SYSTEMS

There are two types of indirect benefits through nonliving systems that were studied the most: avoided materials damages, and improved visibility. The steps of estimating these benefits are summarized in the sections that follow.

5.1 AVOIDED MATERIALS DAMAGES

Anthropogenic sulfur and nitrogen pollutants are believed to have caused vast damage to buildings, structures, as well as the cultural heritage in the past century. Much of the damage occurred in Europe and North America, but with growing car traffic, and very high concentrations of sulfur dioxide in many cities of China, India, and Latin America material damages due to air pollution continue to remain a significant problem.

Objects most susceptible to air pollution are the ones with long lives, particularly buildings. Other objects, such as cars, may be damaged by air pollution, but these damages tend to be less important, because they are usually replaced before the damage could become significant.

Pollutants that contribute to degradation of buildings are particles (particularly soon) causing soiling, and sulfur dioxide (SO_2) contributing to corrosion and erosion caused by acid rain.

Principal effects associated with air pollution are:

- loss of mechanical strength
- leakage,
- failure of protective coatings,
- loss of details in carvings,
- pipe corrosion.

SO_2 has a strong accelerating effect on the degradation of certain materials by contributing to corrosion by acidic deposition. Atmospheric corrosion is influenced by climatic patterns such as relative humidity, temperature and precipitation, and it tends to be a local problem, because the damage often occurs near the source of emission. On the other hand, indirect effects of SO_2 emissions caused the acidification of soil and water bodies, tend to be a regional problem due to the long-range transport of air pollutants.

Air pollution damages materials such as zinc, copper, stone, as well as organic materials. In case of zinc and copper, the dissolution of protective corrosion products leads to increased deterioration rates. Calcareous stones, such as limestone or marble, are very susceptible to acid deposition by sulfur dioxide through transformation of the original calcium carbonate to gypsum and calcium sulfate. Degradation of organic materials, such

as rubber tires and paints, are usually associated with ozone in conjunction with temperature and solar radiation.

5.1.1 Quantifying materials damages

The ideal approach of quantifying materials damages is analogous to the approach used to quantify health endpoints. One would start by estimating the change in pollutant concentrations caused by a policy-induced reduction in emissions. The second step would involve the use of dose-response or concentration-response (CR) functions that relate the physical damage to ambient pollutant concentrations. And the last step involves attaching monetary values to damages.

A valid CR function provides a mathematical relationship between properties of the environment and some index of materials, such as loss of stock thickness. Some early attempts aimed at deriving such CR functions focused on the relationship between ambient pollutant concentrations and corrosion rates. As Lipfert (1996) points out, this approach neglects the important variable of delivery of the reactant to the surface. A full understanding of the process requires a separation of pollutant delivery process from the subsequent chemical reactions. The appropriate technique to estimate a CR function is that of a multiple regression, in which some index of corrosion is the independent variable and the various environmental factors are the independent variables.

Perhaps the most difficult element of economic assessment of materials damages (or benefits from reduced air pollution) is the problem of estimating stocks of buildings at risk (Lipfert and Daum, 1992). One problem is that there is considerable heterogeneity in the use of housing materials across country. While residential housing materials tend to follow regional patterns, commercial and industrial buildings tend to be more uniform. Another important problem since atmospheric corrosion has been present in many parts of the country for a long time, people may have substituted away from the more sensitive building materials toward less sensitive ones. The greatest difficulty lies in distinguishing chemical characteristics of exposed surfaces within each building type and category. Lipfert (1996) suggests that there is a need for a probabilistic, as opposed to deterministic, approach to assessment. There are many relevant but disparate databases on building stocks, but no effort has been made yet to synthesize that information (Lipfert, 1996).

As an alternative to the above bottom-up approach, Rabl (1999) estimates damages to buildings in France by working with aggregate data on observed frequencies of cleaning and repair activities. The result is a "combined concentration-response function". The main variables in the CR function are income and the ambient concentration of particulate matter.

Rabl (1999) considers two types of damage caused by air pollution:

- Corrosion or erosion of coatings and contruction materials

- Soiling

Corrosion and erosion are primarily due to acid deposition. A large number of studies analyzed the effects of air pollution and corrosion and erosion. Dose-response functions have been estimated for several building materials (e.g., Kucera, 1990; Haneef et al., 1992; Butlin et al., 1992, Lipfert, 1987). There are relatively few studies on soiling due to air pollution, and consequently few dose-response functions are available (Hamilton and Mansfield, 1992).

5.1.2 Valuation of materials damages

Given a valid dose response function, using the bottom-up approach one would estimate the repair cost due to air pollution as follows:

Total Repair Cost = Sum Surface Area * Repair Frequency * Repair Cost

Using a bottom-up approach the following are the steps in valuations:

- Division into pollution strata
- Materials inventory and inspection of physical damage
- Damage functions
- Estimated change in service life
- Maintenance/ Replacement cost
- Estimated economic damage

The main drawback of the bottom-approach is the need for very detailed data on building inventories. As an alternative to the bottom-up approach, Rabl (1999) uses a linear regression of renovation expenditures against income, PM_{13}, and SO_2. The best regression model was the following:

$$R = \beta_0 + \beta_1 Income + \beta_2 PM_{13}$$

where R and Income are measured in monetary units per person per year, while, PM_{13} is the measure of particulate matter concentrations in $\mu g / m^3$, and β_0, β_1 and β_2 are parameters to be estimated from the data. The above equation is what Rabl (1999) calls a combined or aggregate concentration-response function. The change in repair costs in response to a change in pollutant concentrations is then given by the following expression:

$$\frac{\partial R}{\partial PM_{13}} = \beta_2 Income$$

Neither approach to valuation may be used to assess damages to the cultural heritage. Cultural heritage encompasses both outdoor buildings and sculptures and treasured objects kept indoors, stored in museums and archives. The most appropriate valuation method for assessment is contingent valuation. These valuation studies tend to be case specific and generally not transferable.

5.2 VISIBILITY BENEFITS

Reduced visibility due to anthropogenic air pollution affects some of the country's most scenic areas. US EPA estimates that in national parks in the eastern United States, average visual range has decreased from 90 miles to 15-25 miles. In the West, visual range has decreased from 140 miles to 35-90 miles. The main cause of visibility impairment is haze. Under stagnant air mass conditions, aerosols can be trapped and produce a visibility condition usually referred to as layered haze. Some light is absorbed by particles while other light may be scattered away before it reaches the observer. The introduction of particulate matter and certain gases into the atmosphere therefore reduces visibility.

From a technical point of view, visibility is a complex and difficult concept to define. Visibility includes psychophysical processes and concurrent value judgments of visual impacts, as well as the physical interaction of light with particles in the atmosphere. Therefore it is important to understand the psychological process involved in viewing a scenic resource, and to be able to establish a link between the physical and psychological processes.

5.2.1 Quantifying visibility benefits

Quantifying visibility requires developing links between visibility and particles that scatter and absorb light. Visibility, in the most general sense, reduces to understanding the effect that various types of aerosol and lighting conditions have on the appearance of landscape features. Measuring visibility by a single index is, however, not possible because visibility cannot be defined by a single parameter (Malm, 1999). Many visibility indices have been proposed, however the most simple and direct way of communicating reduced visibility is through a photograph. In fact, many contingent valuation (CV) studies of visibility present the subjects with photographs of scenic areas with varying levels of visibility. The reason photographs communicate visibility changes so well is that the human eye works much like a camera. The human eye detects relative differences in brightness rather than the overall brightness level, that is to say, the eye measures contrast between adjacent objects.

Because the human eye function like a camera, a photograph captures visibility changes, as humans perceive it. However, it is difficult to extract quantitative information from photographs, and therefore direct measure of fundamental optical measures of the atmosphere have been developed. The most common measures are atmospheric extinction and scattering.

The scattering coefficient is a measure of the ability of particles to scatter photons out of a beam of light, while the absorption coefficient is a measure of how many photons are absorbed. Both coefficients are expressed as a number proportional to the amount of photons scattered or absorbed per distance. The sum of scattering and absorption is referred to as extinction or attenuation.

5.2.2 Valuation of visibility benefits

The most commonly used methods for visibility valuation are hedonic property values and contingent valuation. Hedonic methods are based on revealed preference of consumers, because they link nonmarket valuation to a traded commodity. Hedonic property value studies estimate the marginal WTP function on the basis of an estimated relationship between housing prices and housing attributes (including air quality). There are several factors that affect the relationship between property values and air quality. These include adverse health effects, reduced visibility or soiling due to air pollution. Hedonic methods cannot be used to estimate separately. Disaggregation of overall impacts requires making subjective judgments by the researcher. Nevertheless, the results of hedonic property value studies confirm the hypothesis that air quality has a significant impact on property prices. Kenneth and Greenstone (1998) estimate that the Clean Air Act induced nationwide monetized benefits were $80 billion (in 1982-84 dollars) in the 1970's, and $50 billion during the 1980s. Delucchi, Murphy and McCubbin (2002) estimate monetized costs of total suspended particle pollution in 1990 at $52-$88 billion in (1990 dollars). Some studies (e.g., Brookshire et al., 1979, 1982; Loehman et al., 1994; McLelland et al., 1991) attempted to disaggregate property value impacts into health, visibility, soiling, and other impacts. They find that visibility impacts are the second most important, after health effects, representing 19-34% of total monetized benefits.

Burtraw et al. (1997) present the results of an integrated assessment of the benefits and costs of the Title IV of the 1990 Clean Air Act Amendments initiated reductions in emissions of sulfur dioxide and nitrogen oxides. They use the Tracking and Analysis Framework (TAF) developed for the National Acid Precipitation Assessment Program (NAPAP). Although uncertainties surround their estimates, the findings suggest that the benefits of the program substantially outweigh its costs. Two types of visibility effects are examined: recreational visibility at two national parks (Grand Canyon and Shenandoah), and residential visibility in five metropolitan areas (Albany, NY, Atlantic City, NJ, Charlottesville, VA, Knoxville, TN, and Washington, DC). The results, summarized in Table 5.2.1 below, are most usefully considered on a per capita basis.

Table 5.2.1 Per Capita Benefits in 2010 for Affected Population	
Effect	Benefits per Capita (1990$)
Morbidity	3.50
Mortality	59.29
Aquatic	0.62
Recreational Visibility	3.34
Residential Visibility	5.81
Costs	5.30
Source: Burtraw et al. (1997), Table 2, pp. 13	

These visibility estimates illustrate their potential magnitude, but it should be noted that they are based on relatively small number of studies available in the literature, and also the geographical scope of the project is rather limited. Burtraw et al. (1997) explain the relatively large magnitude of visibility benefits compared to other types of benefits, namely aquatics, by claiming that willingness to pay depends on the availability of substitutes, and visibility, along with health, has no close substitutes.

Smith and Osborne (1996) perform a meta-analysis of visibility valuation studies to test whether CV estimates of WTP are responsive to the amount, or scope, of the environmental amenity being offered. They consider an internal consistency test for CV-based WTP. Internal consistency tests assess the reliability and validity of CV surveys. On way to evaluate the CV method is to compare willingness to pay WTP functions estimated with CV surveys with the specific, observable properties that economic theory implies WTP should follow. Smith and Osborne (1996) selected five of CV studies that used comparable methods for the meta-analysis. These studies focused on air quality as a key element. Furthermore, in each study air quality is presented in a way that permits computation of the change in visible range. The five selected studies are summarized in Table 5.2.2 below.

Table 5.2.2 Summary of CV studies for visibility at national parks analyzed by Smith and Osborne (1996)				
Authors	Mean and inter-quartile range of WTP (per month in 1990 $)	Mean change in visibility	Location	Type of survey
Rowe et al. (1980)	$9.27 ($6.83, $10.82)	0.50	Navaho Recreation Area	In-person interviews administered to to households in area
MacFarland et al	$2.75 ($1.69, $3.73)	1.18	Grand Canyon and Mesa Verde National Parks	In-person interviews administered to visitors to the area
Schulze et al.	$8.50 ($4.42, $11.67)	0.79	Grand Canyon, Mesa Verde, and Zion National Parks	In-person interviews administered to households in Albuquerque, Los Angeles, Denver, and Chicago
Chestnut and Rowe	$4.35 ($3.15, $5.48)	0.62	Grand Canyon, Yosemite, and Shenandoah National Parks	Mail with telephone households in Arizona, Virginia, California, New York, and Missouri
Balson et al.	$0.46 ($0.007, $0.97)	0.955	Grand Canyon National Park	In-person interviews conducted in St. Louis and San Diego Counties
Source: Smith and Osborne (1996), pp. 291, Table 1				

The findings of Smith and Osborne (1996) support a positive, statistically significant and robust relationship between the WTP estimates and the percentage improvement in visible range. These results suggest that it may be possible to transfer results from a meta-analysis of past CV studies. The crucial issue in benefit transfer is to find a common metric to measure the environmental amenity.

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6. SAMPLE CALCULATIONS

The following examples illustrate the steps and calculations involved in the estimation of non-market benefits of the RPS. The examples below focus on mortality and morbidity benefits only.

Step1: Projecting Concentrations

In this step the change in pollutant concentrations around each power plant is estimated. Instead of using a more sophisticated dispersion model, we used the Gaussian Plume Dispersion Model (GPDM) to project pollutant concentrations. This model predicts an average concentration under steady state conditions. The shape of the plume undergoing dispersion is a function of the wind speed, vertical temperature profile and atmospheric stability. GPDM is widely used to predict concentrations in the atmosphere. There are, however, significant simplifications with this model. The main assumptions of GPDM are:

1. Only steady-state concentrations are estimated.
2. Wind blows in x-direction and is constant in both speed and direction
3. Transport with the mean wind is much greater than turbulent transport in the x-direction.
4. The source emission rate (the rate at which the pollutant is emitted per unit of time) is constant.
5. Diffusion coefficients are constant in both time and space.
6. The source emits chemicals of concern (COC) at a point in space x=y=0 and z=H, where H is the effective stack height.
7. The COC's are inert (non-decaying and non-reactive).
8. There is no barrier to plume migration.
9. Mass is conserved across the plume cross section.
10. Mass within a plume follows a Gaussian (normal) distribution in both the crosswind (y) and vertical (z) directions.

The GPDM is derived from the advection-diffusion equation. The general equation to calculate the steady state concentration of an air contaminant in the ambient air resulting from a point source is given by:

$$C(y,x,z) = \frac{Q}{2\pi\pi u_y \sigma_z} \exp\left(\frac{-y^2}{2\sigma_y^2}\right)\left\{\exp\left(\frac{-(z-H)^2}{2\sigma_z^2}\right) + \exp\left(\frac{-(z+H)^2}{2\sigma_z^2}\right)\right\}$$

where

C(x,y,z) = contaminant concentration at the specified coordinate
x = downwind distance

y = crosswind distance
z = vertical distance above ground
Q = contaminant emission rate
σ_y = lateral dispersion coefficient function
σ_z = vertical dispersion coefficient function
u = wind velocity in downwind direction
H = effective stack height

In the above equation σ_y, the lateral dispersion coefficient function, and σ_z, the vertical dispersion coefficient functions depend on the downwind distance and the atmospheric stability class. The value of these coefficients in meters can be obtained from the equations utilized by the Industrial Source Complex (ISC) Dispersion Model developed by USEPA (1995):

$$\sigma_y = 465.11628 \cdot x \cdot \tan(TH)$$

where

$$TH = 0.01745 \cdot [c - d\ln(x)]$$

$$\sigma_z = ax^b$$

A simplified version of the above formula to estimate steady state pollutant concentrations is given by

$$C(y,x,z) = \frac{Q}{2\pi\pi u_y \sigma_z} \exp\left\{-\frac{1}{2}\left(\frac{y^2}{\sigma_y^2} + \frac{(z-H)^2}{\sigma_z^2}\right)\right\}$$

This formula assumes that the pollutant is not reflected from the ground, and therefore it yields lower estimates in general than when one assumes reflection.

EXAMPLE 1: Reduction in excess mortality due to SO_2

Power plant assumptions

We have in mind a coal-burning power plant with the following characteristics:

Capacity = 800MW
Capacity Factor = 0.7[φ]
SO_2 emission factor per output: 10 lbs/MWh
Contaminant emissions rate (g/s): 705
Physical stack height (m): 125
Effective stack height (m): 215.96
Consistent with the followings assumptions:
Stack velocity (m/s): 15
Stack exit diameter (m): 1.5
Stack gas temperature (K): 450
Ambient gas temperature (K): 300
We assume that the RPS results in a 10% reduction in generation by this power plant.

Assumptions about population and health status

Total population: 8.4 million
Total non-accidental deaths: 70,766
Non-accidental mortality per person: 0.00841
Population is assumed to be uniformly distributed in the affected area.
Population density per square kilometer: 402

Meteorological and other assumptions

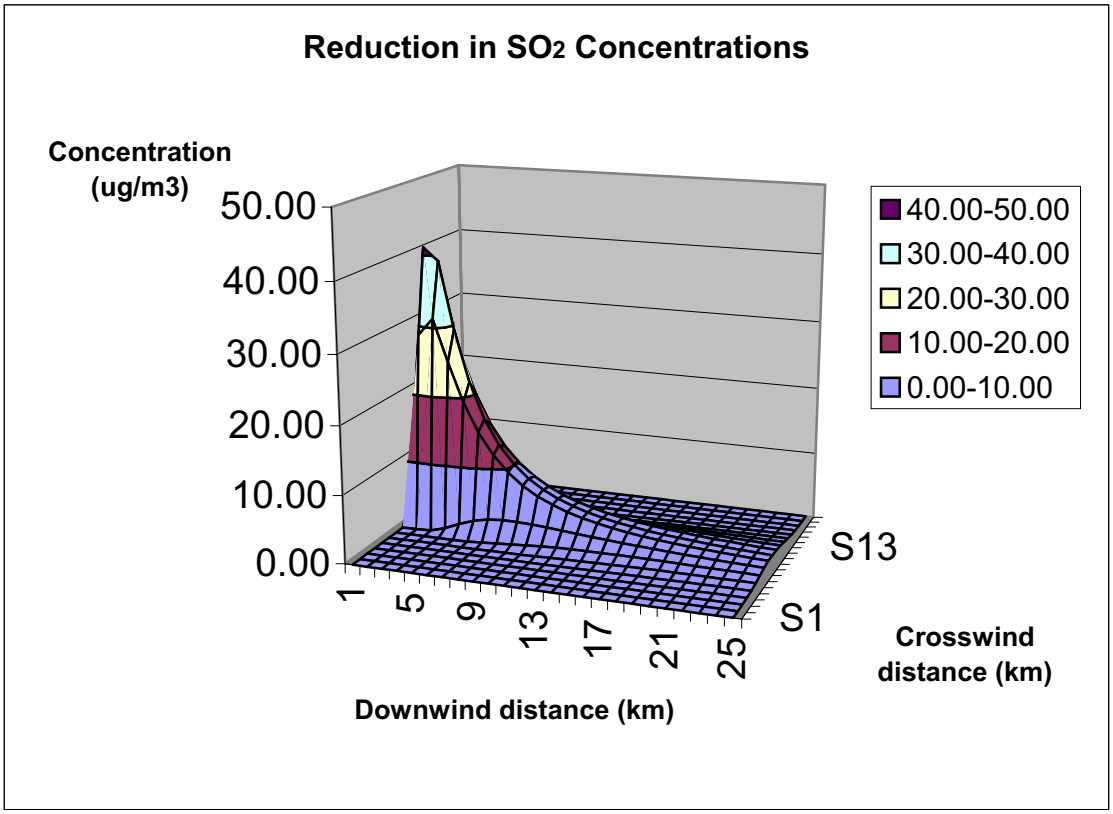
Wind velocity in downward direction (m/s): 1
Incoming solar radiation during the day: moderate
PASQUILL-GIFFORD category: B
Cloud condition at night: mostly overcast
PASQUILL-GIFFORD category: B

Step 2: Quantifying the change in SO_2 mortality

First, using GPDM we calculated SO_2 concentrations for 100-meter grids for 25 km downwind and 9 km crosswind distances. Next, the estimated concentrations were

[φ] Capacity factor is the ratio of the electrical energy produced by a generating unit for the period of time considered to the electrical energy that could have been produced at continuous full power operation during the same period.

averaged at the 1-km grid level. These steps were followed for both the baseline scenario and the policy scenario (10% reduction in generation). The difference in SO_2 concentration between the two scenarios at the 1-km grid level is estimated.



Next concentration response functions are used to calculate the change in non-accidental mortality.

CR-function:

$$\Delta\text{mortality} = -\left(y_1 (e^{\beta \Delta SO_2} - 1) \right) \cdot \text{pop}$$

y_1 = non-accidental deaths per person

β = SO_2 coefficient

ΔSO_2 = change in SO2 concentrations (ppb)

pop = population sample

We used the SO_2 mortality CR function estimated by Touloumi et al. (1996). The 95% confidence interval (CI) for the reduction in non-accidental mortality (statistical deaths) is: (27.5, 72.2)[1] with an estimated value 30.89.

[1] This CI refers only to the uncertainty associated with the CR-function estimation.

Step 3: Monetizing SO_2 mortality benefits

Using the median estimate of Viscusi and Aldy (2003) for the Value of Statistical Life (VSL) of $7 million, the estimated benefit is $216.2 million with a 95% confidence interval of ($192.5m, 505.4m).

EXAMPLE 2: Reduction in excess chronic obstructive pulmonary disease (COPD) hospital admissions due to PM_{10}

This example illustrates the estimation of morbidity benefits associated with improved air quality. The health benefit of interest is avoided hospital admissions for chronic obstructive pulmonary disease (COPD) due to particulate matter 10 μm and less in diameter (PM_{10}). COPD is a group of diseases categorized by ICD-9-CM codes 490-496.

(ICD=International Classification of Diseases)

490 - Bronchitis, not specified as acute or chronic
491 - Chronic bronchitis
492 - Emphysema
493 - Asthma
494 - Bronchiectasis
495 - Extrinsic allergic alveolitis
496 - Chronic airway obstruction, not elsewhere classified

The same power plant, population and meteorological assumptions hold as I the previous example. Morbidity benefits were estimated in the following steps.

Step 1: Determining concentration-response (CR) relationships for COPD admissions.

CR-studies usually assume the following log-linear functional form:

$$\Delta\text{COPD hospital admissions} = -\left(y_1 (e^{\beta PM_{10}} - 1) \right) \cdot \text{pop}$$

y_1 = baseline COPD admission rate, defined as COPD hospital admissions per person
β = estimated PM_{10} coefficient

ΔPM_{10} = change in PM_{10} concentrations ($\mu g/m^3$)
pop = exposed population per km^2

Three studies have been identified that estimated the concentration-response relationship between ΔPM_{10} concentrations and hospital admissions for COPD: Chen at al. (2004) in Vancouver, Canada, Zanobetti et al. (2000) in Chicago, Cook county, IL, and Moolgavkar (2000) in Los Angeles county, CA. The parameter estimates are summarized in the table below.

Study	Parameter estimate	95% Confidence Interval		Study Population	Studied Health Effect
	β	β_L	β_H		ICD-9
Chen et al. (2004)	0.0152463	0.0061760	0.0243167	Ages 65+	490-492,494,496
Zanobetti et al. (2000)	0.0076035	0.0015873	0.0136196	Ages 65+	490-492, 494-496
Moolgavkar (2000)	0.0016073	0.0010603	0.0021542	Ages 0-19	490-496
	0.0007968	0.0002110	0.0016826	Ages 20-64	490-496
	0.0009877	0.0004969	0.0014785	Ages 65+	490-496

Step 2: Determining Baseline Exposure

We use the Gaussian Plume Dispersion Model to predict PM_{10} concentration changes. Under our meteorological assumptions most of the deposition occurs within 25 km in downwind direction and 10 km in crosswind direction. We assume that population is uniformly distributed in the affected area. We use a population density estimate derived from 2003 population estimate figures by the U.S. Census Bureau: 1164 people per square mile, or 450 people per square kilometer. Consequently, the total population that is potentially exposed to PM pollutant from the power plant is approximately 250,000.

Based on U.S. Census Bureau population estimates, we assume the following population density estimate for the various age groups:

Age Group	Share of Total Population	Population Density per Square Kilometer
0-19	27.2%	122.1
20-64	59.7%	268.3
65+	13.2%	59.4

Step 3: Determining the Number of Baseline Cases for Each Quantifiable Health Effect Number exposed x Baseline exposure x Dose-response relationship.

Because the health effects studied in the three studies are not identical, it was necessary to estimate and make assumptions about the baseline hospital admission rate for each group of health effects (ICD codes). The Healthcare Cost & Utilization Project (HCUP) by the Agency for Healthcare Research and Quality's (AHRQ) (http://www.ahrq.gov/hcupnet/), estimated the number of hospital discharges for the various COPD conditions by various characteristics, such as age, sex, income, etc., at the national and regional (but not state) level. Because our CR-functions are estimated for various age groups, it was necessary to estimate the hospital admission rate for each age group. Hospital admissions were estimated for the following age groups from the 2002 national data:

Age Group	Share of Total Population	ICD 490-492, 494,496	ICD 490-492, 494-496	ICD 490-496
0-17	25.3%	0.00006174	0.00006174	0.0020353
18-64	62.3%	0.00112073	0.00112073	0.0021734
65+	12.4%	0.0116307	0.0116307	0.0136903

Step 4: Determining Exposure for each Policy Scenario, Determining the Number of Cases for Each Quantifiable Effect with the Regulation, Determining the Number of Cases Avoided as a Result of Each Regulatory Option

The avoided COPD hospital admissions for the various age groups and CR functions and dispersion models are summarized in the tables below.

Dispersion Model: Gaussian Plume Dispersion without Reflection					
Estimated Reduction in COPD Hospital Admissions	95% Confidence Interval		CR-Function	Population	Location
6.28	2.40	10.66	Chen et al. (2004)	Ages 65+	Vancouver, Canada
2.98	0.60	5.55	Zanobetti et al. (2000)	Ages 65+ (Medicare admissions)	Chicago, Cook County, IL
0.44	0.22	0.66	Moolgavkar (2000)	Ages 65+	Los Angeles County
0.25	0.07	0.54	Moolgavkar (2000)	Ages 20-64	Los Angeles County
0.22	0.14	0.29	Moolgavkar (2000)	Ages 0-19	Los Angeles County

Dispersion Model: Gaussian Plume Dispersion with Reflection					
Estimated Reduction in COPD Hospital Admissions	95% Confidence Interval		CR-Function	Population	Location
15.81	5.68	28.88	Chen et al. (2004)	Ages 65+	Vancouver, Canada

7.12	1.38	13.81	Zanobetti et al. (2000)	Ages 65+	Chicago, Cook County, IL
1.00	0.50	1.51	Moolgavkar (2000)	Ages 65+	Los Angeles County
0.58	0.15	1.24	Moolgavkar (2000)	Ages 20-64	Los Angeles County
0.50	0.33	0.68	Moolgavkar (2000)	Ages 0-19	Los Angeles County

Step 5: Monetizing the health benefits

Benefits were monetized using the cost-of-illness approach. Using HCUP estimates of mean cost of hospital admissions, weighted cost was estimated for each age group and each groups of ICD codes. The following value were used to monetize benefits:

Age Group	ICD-9 Codes	Average Cost ($2002)
Ages 65+	490-496	15,537.05
Ages 65+	490-492, 494-496	13,908.82
Ages 65+	490-492, 494, 496	13,886.41
Ages 19-64	490-496	12,421.90
Ages 0-18	490-496	7,511.36

Monetized benefits for the two dispersion models are summarized in the tables below.

Dispersion without reflection	Population	Avoided COPD Admissions (annual)	Monetary Value ($2002)	95% Confidence interval	
Chen et al. (2004)	65+	6.3	87,186.3	33,319.7	147,969.3
Zanobetti et al. (2000)	65+ Medicare patients	3.0	41,455.3	8,340.3	77,177.5
Moolgavkar (2000)	All ages	0.9	11,583.5	5,319.3	19,087.0

Dispersion with relfection	Population	Avoided COPD Admissions (annual)	Monetary Value ($2002)	95% Confidence interval	
Chen et al. (2004)	65+	15.8	219,559.5	78,847.9	401,042.0
Zanobetti et al. (2000)	65+ Medicare patients	7.1	98,996.9	19,198.5	192,047.0
Moolgavkar (2000)	All ages	2.1	26,578.3	12,173.5	43,946.7

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