

Air Toxics Analysis in New Jersey: Ambient Data Review and Model Validation

Final Report

**Prepared by
Northeast States for Coordinated Air Use Management
(NESCAUM)**

June 4, 2012

Members of Northeast States for Coordinated Air Use Management

Arthur Marin, Executive Director
Northeast States for Coordinated Air Use Management

Anne Gobin, Bureau Chief
Connecticut Department of Energy & Environmental Protection, Bureau of Air
Management

Melanie Loyzim, Bureau Director
Maine Department of Environmental Protection, Bureau of Air Quality

Nancy L. Seidman, Deputy Assistant Commissioner
Massachusetts Department of Environmental Protection, Bureau of Waste Prevention

Craig Wright, Acting Director
New Hampshire Department of Environmental Services, Air Resources Division

William O'Sullivan, Director
New Jersey Department of Environmental Protection, Office of Air Quality Management

David Shaw, Director
New York Department of Environmental Conservation, Division of Air Resources

Douglas L. McVay, Chief
Rhode Island Department of Environmental Management, Office of Air Resources

Richard A. Valentinetti, Director
Vermont Department of Environmental Conservation, Air Pollution Control Division

Air Toxics Analysis in New Jersey: Ambient Data Review and Model Validation

Prepared by
Northeast States for Coordinated Air Use Management
(NESCAUM)

June 4, 2012

AIR TOXICS ANALYSIS IN NEW JERSEY: AMBIENT DATA REVIEW AND MODEL VALIDATION

FINAL REPORT

Project Managers

Alexander Polissar, NJDEP
Paul Miller, NESCAUM

Principal Contributors

Leiran Biton, NESCAUM
Kathleen Fahey, NESCAUM (now at U.S. EPA)
Laura Shields, NESCAUM
Huiyan Yang, NESCAUM (now at the University of Texas at El Paso)

TABLE OF CONTENTS

Executive Summary	xvii
1. Introduction.....	1-1
1.1. Overview of air toxics.....	1-1
1.2. Air toxics in New Jersey	1-2
1.3. Previous receptor modeling analysis	1-3
1.4. Air toxics monitoring.....	1-4
1.5. Air quality modeling	1-5
1.6. Layout of this report.....	1-6
2. Analysis of the National Air Toxics Assessment (NATA).....	2-1
2.1. About NATA 2005	2-2
2.2. Results from NATA 2005	2-4
2.3. Sources, atmospheric processes, and health effects of risk-driving compounds	2-9
2.4. Grouping air toxic species	2-17
2.5. Comparison of NATA data with monitoring data	2-17
3. Analysis of air toxics monitoring data	3-1
3.1. Air toxics monitoring networks	3-2
3.1.1. New Jersey Air Toxics network.....	3-3
3.1.2. Photochemical Assessment Monitoring Stations (PAMS)	3-3
3.1.3. Measurements from neighboring states	3-3
3.2. Monitoring data sources and processing.....	3-4
3.3. Sampling methods and uncertainty	3-7
3.4. Monitoring data results	3-8
3.4.1. Summary of New Jersey Air Toxics monitoring data results	3-8
3.4.2. Summary of New Jersey PAMS data results	3-9
3.4.3. Summary of PAMS and NJ Air Toxics data comparisons	3-9
3.4.4. Summary of regional air toxics monitoring data	3-10
3.4.5. Detailed monitoring data results	3-11
4. Photochemical air toxics model evaluation	4-1
4.1. Modeling air toxics	4-1
4.1.1. Types of models: strengths and weaknesses	4-2
4.1.2. Model uncertainty and sensitivity.....	4-3
4.2. Observational data	4-4
4.3. Model evaluation methodology and discussion.....	4-4
4.4. CMAQ model settings	4-8
4.4.1. Domain.....	4-8
4.4.2. Meteorology.....	4-8
4.4.3. Emissions	4-9
4.4.4. Boundary conditions and initial conditions	4-9
4.5. Model evaluation results	4-10
4.5.1. Regional haze model evaluation results.....	4-11
4.5.2. Air toxics model evaluation results.....	4-44

4.6. Comparison of the regional haze and air toxics models	4-74
4.7. Conclusions about the models	4-75
5. Summary	5-1
6. References	6-1
Appendix A: Comparison of photochemical assessment monitoring stations and New Jersey air toxics site data	A-1
Appendix B: Regional air toxics measurements	B-1

FIGURES

Figure ES-1. Air toxics monitoring sites in New Jersey.....	xxi
Figure 2-1. Top five pollutants contributing to New Jersey cancer risk by source category	2-6
Figure 2-2. Top five pollutants contributing to New Jersey respiratory hazard index by source category.....	2-7
Figure 2-3. Top five pollutants contributing to New Jersey neurological hazard index by source category.....	2-9
Figure 2-4. Comparison of NATA 2002 predicted and observed concentrations at Camden (Camden County).....	2-18
Figure 2-5. Comparison of NATA 2002 predicted and observed concentrations at Chester (Morris County)	2-18
Figure 2-6. Comparison of NATA 2002 predicted and observed concentrations at Elizabeth (Union County)	2-19
Figure 2-7. Comparison of NATA 2002 predicted and observed concentrations at New Brunswick (Middlesex County).....	2-19
Figure 3-1. Air toxics monitoring sites in New Jersey	3-1
Figure 3-2. Air toxics monitoring sites in states neighboring New Jersey	3-4
Figure 3-3. Quarterly and annual average benzene concentrations for 2000-2008 at Camden.....	3-13
Figure 3-4. Quarterly and annual average benzene concentrations for 2001-2009 at Chester.....	3-13
Figure 3-5. Quarterly and annual average benzene concentrations for 2000-2009 at Elizabeth.....	3-14
Figure 3-6. Quarterly and annual average benzene concentrations for 2000-2009 at New Brunswick	3-14
Figure 3-7. Summer and winter average benzene concentrations at Camden, Chester, Elizabeth, and New Brunswick.....	3-15
Figure 3-8. Comparison of annual average benzene concentrations by site.....	3-15
Figure 3-9. Comparison of average hourly benzene concentrations for 2000-2008 at the Camden PAMS.....	3-16
Figure 3-10. Comparison of average hourly benzene concentrations for 2000-2008 at the Rutgers University PAMS.....	3-17
Figure 3-11. Comparison of average hourly benzene concentrations for 2000-2008 at the Rider University PAMS	3-18
Figure 3-12. Quarterly and annual average 1,3-butadiene concentrations for 2000-2008 at Camden	3-19
Figure 3-13. Quarterly and annual average 1,3-butadiene concentrations for 2001-2009 at Chester	3-20
Figure 3-14. Quarterly and annual average 1,3-butadiene concentrations for 2000-2009 at Elizabeth	3-20
Figure 3-15. Quarterly and annual average 1,3-butadiene concentrations for 2000-2009 at New Brunswick.....	3-21
Figure 3-16. Summer and winter average 1,3-butadiene concentrations at Camden, Chester, Elizabeth, and New Brunswick	3-21
Figure 3-17. Comparison of annual average 1,3-butadiene concentrations by site	3-22

Figure 3-18. Quarterly and annual average xylenes concentrations for 2000-2008 at Camden.....	3-24
Figure 3-19. Quarterly and annual average xylenes concentrations for 2001-2009 at Chester.....	3-25
Figure 3-20. Quarterly and annual average xylenes concentrations for 2000-2009 at Elizabeth.....	3-26
Figure 3-21. Quarterly and annual average xylenes concentrations for 2000-2009 at New Brunswick.....	3-27
Figure 3-22. Summer and winter average xylenes concentrations at Camden, Chester, Elizabeth, and New Brunswick.....	3-28
Figure 3-23. Comparison of annual average xylenes concentrations by site.....	3-28
Figure 3-24. Comparison of average hourly <i>o</i> -xylene isomer concentrations for 2000-2008 at the Camden PAMS.....	3-30
Figure 3-25. Comparison of average hourly <i>m,p</i> -xylene isomers concentrations for 2000-2008 at the Camden PAMS.....	3-31
Figure 3-26. Comparison of average hourly <i>o</i> -xylene isomer concentrations for 2000-2008 at the Rutgers University PAMS.....	3-32
Figure 3-27. Comparison of average hourly <i>m,p</i> -xylene isomers concentrations for 2000-2008 at the Rutgers University PAMS.....	3-33
Figure 3-28. Comparison of average hourly <i>o</i> -xylene isomer concentrations for 2000-2008 at the Rider University PAMS.....	3-34
Figure 3-29. Comparison of average hourly <i>m,p</i> -xylene isomers concentrations for 2000-2008 at the Rider University PAMS.....	3-35
Figure 3-30. Quarterly and annual average toluene concentrations for 2000-2008 at Camden.....	3-37
Figure 3-31. Quarterly and annual average toluene concentrations for 2001-2009 at Chester.....	3-37
Figure 3-32. Quarterly and annual average toluene concentrations for 2000-2009 at Elizabeth.....	3-38
Figure 3-33. Quarterly and annual average toluene concentrations for 2000-2009 at New Brunswick.....	3-38
Figure 3-34. Summer and winter average toluene concentrations at Camden, Chester, Elizabeth, and New Brunswick.....	3-39
Figure 3-35. Comparison of annual average toluene concentrations by site.....	3-39
Figure 3-36. Comparison of average hourly toluene concentrations for 2000-2008 at the Camden PAMS.....	3-40
Figure 3-37. Comparison of average hourly toluene concentrations for 2000-2008 at the Rutgers University PAMS.....	3-41
Figure 3-38. Comparison of average hourly toluene concentrations for 2000-2008 at the Rider University PAMS.....	3-42
Figure 3-39. Quarterly and annual average ethylbenzene concentrations for 2000- 2008 at Camden.....	3-43
Figure 3-40. Quarterly and annual average ethylbenzene concentrations for 2001- 2009 at Chester.....	3-44
Figure 3-41. Quarterly and annual average ethylbenzene concentrations for 2000- 2009 at Elizabeth.....	3-44

Figure 3-42. Quarterly and annual average ethylbenzene concentrations for 2000-2009 at New Brunswick	3-45
Figure 3-43. Summer and winter average ethylbenzene concentrations at Camden, Chester, Elizabeth, and New Brunswick	3-45
Figure 3-44. Comparison of annual average ethylbenzene concentrations by site.....	3-46
Figure 3-45. Comparison of average hourly ethylbenzene concentrations for 2000-2008 at the Camden PAMS	3-47
Figure 3-46. Comparison of average hourly ethylbenzene concentrations for 2000-2008 at the Rutgers University PAMS	3-48
Figure 3-47. Comparison of average hourly ethylbenzene concentrations for 2000-2008 at the Rider University PAMS	3-49
Figure 3-48. Quarterly and annual average carbon tetrachloride concentrations for 2000-2008 at Camden.....	3-51
Figure 3-49. Quarterly and annual average carbon tetrachloride concentrations for 2001-2009 at Chester.....	3-52
Figure 3-50. Quarterly and annual average carbon tetrachloride concentrations for 2000-2009 at Elizabeth.....	3-52
Figure 3-51. Quarterly and annual average carbon tetrachloride concentrations for 2000-2009 at New Brunswick.....	3-53
Figure 3-52. Summer and winter average carbon tetrachloride concentrations at Camden, Chester, Elizabeth, and New Brunswick	3-53
Figure 3-53. Comparison of annual average carbon tetrachloride concentrations by site	3-54
Figure 3-54. Quarterly and annual average formaldehyde concentrations for 2000-2008 at Camden	3-55
Figure 3-55. Quarterly and annual average formaldehyde concentrations for 2001-2009 at Chester	3-56
Figure 3-56. Quarterly and annual average formaldehyde concentrations for 2000-2009 at Elizabeth	3-56
Figure 3-57. Quarterly and annual average formaldehyde concentrations for 2000-2009 at New Brunswick.....	3-57
Figure 3-58. Summer and winter average formaldehyde concentrations at Camden, Chester, Elizabeth, and New Brunswick	3-58
Figure 3-59. Comparison of annual average formaldehyde concentrations by site.....	3-58
Figure 3-60. Comparison of weekday and weekend average 3-hour formaldehyde concentrations for 2000, 2001, 2003, and 2005 at the Camden PAMS	3-59
Figure 3-61. Quarterly and annual average acetaldehyde concentrations for 2000-2008 at Camden.....	3-61
Figure 3-62. Quarterly and annual average acetaldehyde concentrations for 2001-2009 at Chester.....	3-61
Figure 3-63. Quarterly and annual average acetaldehyde concentrations for 2000-2009 at Elizabeth.....	3-62
Figure 3-64. Quarterly and annual average acetaldehyde concentrations for 2000-2009 at New Brunswick	3-62
Figure 3-65. Summer and winter average acetaldehyde concentrations at Camden, Chester, Elizabeth, and New Brunswick	3-63

Figure 3-66. Comparison of annual average acetaldehyde concentrations by site	3-63
Figure 3-67. Comparison of weekday and weekend average 3-hour acetaldehyde concentrations for 2000, 2001, 2003, and 2005 at the Camden PAMS	3-64
Figure 3-68. Quarterly and annual average acrolein concentrations for 2005-2008 at Camden.....	3-65
Figure 3-69. Quarterly and annual average acrolein concentrations for 2005-2009 at Chester.....	3-65
Figure 3-70. Quarterly and annual average acrolein concentrations for 2005-2009 at Elizabeth.....	3-66
Figure 3-71. Quarterly and annual average acrolein concentrations for 2005-2009 at New Brunswick	3-66
Figure 3-72. Summer and winter average acrolein concentrations at Camden, Chester, Elizabeth, and New Brunswick.....	3-67
Figure 3-73. Comparison of annual average acrolein concentrations by site	3-67
Figure 4-1. CMAQ modeling domains	4-9
Figure 4-2. Comparison of monthly average higher aldehyde concentrations for the NJ Air Toxics network at all sites for the regional haze model	4-19
Figure 4-3. Comparison of monthly average formaldehyde concentrations for the NJ Air Toxics network at all sites for the regional haze model	4-20
Figure 4-4. Comparison of monthly average olefin concentrations for the NJ Air Toxics network at all sites for the regional haze model.....	4-21
Figure 4-5. Comparison of monthly average paraffin concentrations for the NJ Air Toxics network at all sites for the regional haze model	4-22
Figure 4-6. Comparison of monthly average toluene concentrations for the NJ Air Toxics network at all sites for the regional haze model.....	4-23
Figure 4-7. Comparison of monthly average xylenes concentrations for the NJ Air Toxics network at all sites for the regional haze model	4-24
Figure 4-8. Comparison of July diurnal patterns for higher aldehydes from the PAMS network at all sites for the regional haze model.....	4-25
Figure 4-9. Comparison of July diurnal patterns for ethylene from the PAMS network at all sites for the regional haze model.....	4-26
Figure 4-10. Comparison of July diurnal patterns for isoprene from the PAMS network at all sites for the regional haze model.....	4-27
Figure 4-11. Comparison of July diurnal patterns for olefins from the PAMS network at all sites for the regional haze model.....	4-28
Figure 4-12. Comparison of July diurnal patterns for paraffins from the PAMS network at all sites for the regional haze model.....	4-29
Figure 4-13. Comparison of July diurnal patterns for toluene from the PAMS network at all sites for the regional haze model.....	4-30
Figure 4-14. Comparison of July diurnal patterns for xylenes from the PAMS network at all sites for the regional haze model.....	4-31
Figure 4-15. Comparison of January scatter plot for higher aldehydes from NJ Air Toxics sites for the regional haze model	4-33
Figure 4-16. Comparison of January scatter plot for formaldehyde from NJ Air Toxics sites for the regional haze model.....	4-34

Figure 4-17. Comparison of January scatter plot for olefins from NJ Air Toxics sites for the regional haze model.....	4-35
Figure 4-18. Comparison of January scatter plot for paraffins from NJ Air Toxics sites for the regional haze model.....	4-36
Figure 4-19. Comparison of January scatter plot for toluene from NJ Air Toxics sites for the regional haze model.....	4-37
Figure 4-20. Comparison of January scatter plot for xylenes from NJ Air Toxics sites for the regional haze model.....	4-38
Figure 4-21. Comparison of July scatter plot for higher aldehydes from NJ Air Toxics sites for the regional haze model.....	4-39
Figure 4-22. Comparison of July scatter plot for formaldehyde from NJ Air Toxics sites for the regional haze model.....	4-40
Figure 4-23. Comparison of July scatter plot for olefins from NJ Air Toxics sites for the regional haze model.....	4-41
Figure 4-24. Comparison of July scatter plot for paraffins from NJ Air Toxics sites for the regional haze model.....	4-42
Figure 4-25. Comparison of July scatter plot for toluene from NJ Air Toxics sites for the regional haze model.....	4-43
Figure 4-26. Comparison of July scatter plot for xylenes from NJ Air Toxics sites for the regional haze model.....	4-44
Figure 4-27. Comparison of monthly average higher aldehyde concentrations for the NJ Air Toxics network at all sites for the air toxics model.....	4-49
Figure 4-28. Comparison of monthly average formaldehyde concentrations for the NJ Air Toxics network at all sites for the air toxics model.....	4-50
Figure 4-29. Comparison of monthly average olefin concentrations for the NJ Air Toxics network at all sites for the air toxics model	4-51
Figure 4-30. Comparison of monthly average paraffin concentrations for the NJ Air Toxics network at all sites for the air toxics model	4-52
Figure 4-31. Comparison of monthly average toluene concentrations for the NJ Air Toxics network at all sites for the air toxics model	4-53
Figure 4-32. Comparison of monthly average xylenes concentrations for the NJ Air Toxics network at all sites for the air toxics model	4-54
Figure 4-33. Comparison of July diurnal patterns for higher aldehydes from the PAMS network at all sites for the air toxics model	4-55
Figure 4-34. Comparison of July diurnal patterns for ethylene from the PAMS network at all sites for the air toxics model	4-56
Figure 4-35. Comparison of July diurnal patterns for isoprene from the PAMS network at all sites for the air toxics model	4-57
Figure 4-36. Comparison of July diurnal patterns for olefins from the PAMS network at all sites for the air toxics model	4-58
Figure 4-37. Comparison of July diurnal patterns for paraffins from the PAMS network at all sites for the air toxics model	4-59
Figure 4-38. Comparison of July diurnal patterns for toluene from the PAMS network at all sites for the air toxics model	4-60
Figure 4-39. Comparison of July diurnal patterns for xylenes from the PAMS network at all sites for the air toxics model	4-61

Figure 4-40. Comparison of January scatter plot for higher aldehydes from NJ Air Toxics sites for the air toxics model	4-63
Figure 4-41. Comparison of January scatter plot for formaldehyde from NJ Air Toxics sites for the air toxics model	4-64
Figure 4-42. Comparison of January scatter plot for olefins from NJ Air Toxics sites for the air toxics model	4-65
Figure 4-43. Comparison of January scatter plot for paraffins from NJ Air Toxics sites for the air toxics model	4-66
Figure 4-44. Comparison of January scatter plot for toluene from NJ Air Toxics sites for the air toxics model	4-67
Figure 4-45. Comparison of January scatter plot for xylenes from NJ Air Toxics sites for the air toxics model	4-68
Figure 4-46. Comparison of July scatter plot for higher aldehydes from NJ Air Toxics sites for the air toxics model	4-69
Figure 4-47. Comparison of July scatter plot for formaldehyde from NJ Air Toxics sites for the air toxics model	4-70
Figure 4-48. Comparison of July scatter plot for olefins from NJ Air Toxics sites for the air toxics model	4-71
Figure 4-49. Comparison of July scatter plot for paraffins from NJ Air Toxics sites for the air toxics model	4-72
Figure 4-50. Comparison of July scatter plot for toluene from NJ Air Toxics sites for the air toxics model	4-73
Figure 4-51. Comparison of July scatter plot for xylenes from NJ Air Toxics sites for the air toxics model	4-74
Figure A-1. Comparison of averaged PAMS and NJ Air Toxics measurements for formaldehyde at Camden	A-3
Figure A-2. Comparison of averaged PAMS and NJ Air Toxics measurements for acetaldehyde at Camden.....	A-3
Figure A-3. Comparison of averaged PAMS and NJ Air Toxics measurements for benzene at Camden	A-4
Figure A-4. Scatter plot of averaged PAMS and NJ Air Toxics measurements for benzene at Camden	A-5
Figure A-5. Summer average PAMS and NJ Air Toxics measurements for benzene at Camden for 2000-2008	A-6
Figure A-6. Comparison of averaged PAMS and NJ Air Toxics measurements for xylenes at Camden	A-7
Figure A-7. Scatter plot of averaged PAMS and NJ Air Toxics measurements for xylenes at Camden	A-8
Figure A-8. Summer average PAMS and NJ Air Toxics measurements for xylenes at Camden for 2000-2008	A-9
Figure A-9. Comparison of averaged PAMS and NJ Air Toxics measurements for toluene at Camden.....	A-10
Figure A-10. Scatter plot of averaged PAMS and NJ Air Toxics measurements for toluene at Camden	A-11
Figure A-11. Summer average PAMS and NJ Air Toxics measurements for toluene at Camden for 2000-2008.....	A-12

Figure A-12. Comparison of averaged PAMS and NJ Air Toxics measurements for ethylbenzene at Camden.....	A-13
Figure A-13. Scatter plot of averaged PAMS and NJ Air Toxics measurements for ethylbenzene at Camden.....	A-14
Figure A-14. Summer average PAMS and NJ Air Toxics measurements for ethylbenzene at Camden for 2000-2008	A-15
Figure B-1. Quarterly and annual average benzene concentrations at Sherwood Island, Connecticut.....	B-2
Figure B-2. Quarterly and annual average benzene concentrations at Botanical Gardens, NY.....	B-3
Figure B-3. Quarterly and annual average benzene concentrations at Queens Community College, New York	B-4
Figure B-4. Quarterly and annual average benzene concentrations at East Lycoming, Pennsylvania.....	B-5
Figure B-5. Quarterly and annual average benzene concentrations at Lums Pond, Delaware	B-6
Figure B-6. Comparison of average hourly benzene concentrations at the Sherwood Island, Connecticut PAMS	B-7
Figure B-7. Comparison of average hourly benzene concentrations at the Botanical Gardens, New York PAMS	B-7
Figure B-8. Comparison of average hourly benzene concentrations at the Queens Community College, New York PAMS	B-8
Figure B-9. Comparison of average hourly benzene concentrations at the East Lycoming, Pennsylvania PAMS	B-8
Figure B-10. Comparison of average hourly benzene concentrations at the Lums Pond, Delaware PAMS	B-9
Figure B-11. Quarterly and annual average <i>m,p</i> -xylene concentrations at Sherwood Island, Connecticut	B-10
Figure B-12. Quarterly and annual average <i>m,p</i> -xylene concentrations at Botanical Gardens, New York	B-11
Figure B-13. Quarterly and annual average <i>m,p</i> -xylene concentrations at Queens Community College, New York	B-12
Figure B-14. Quarterly and annual average <i>m,p</i> -xylene concentrations at East Lycoming, Pennsylvania.....	B-12
Figure B-15. Quarterly and annual average <i>m,p</i> -xylene concentrations at Lums Pond, Delaware	B-13
Figure B-16. Comparison of average hourly <i>m,p</i> -xylene concentrations at the Sherwood Island, Connecticut PAMS	B-13
Figure B-17. Comparison of average hourly <i>m,p</i> -xylene concentrations at the Botanical Gardens, New York PAMS	B-14
Figure B-18. Comparison of average hourly <i>m,p</i> -xylene concentrations at the Queens Community College, New York PAMS	B-14
Figure B-19. Comparison of average hourly <i>m,p</i> -xylene concentrations at the East Lycoming, Pennsylvania PAMS	B-15
Figure B-20. Comparison of average hourly <i>m,p</i> -xylene concentrations at the Lums Pond, Delaware PAMS	B-15

Figure B-21. Quarterly and annual average toluene concentrations at Sherwood Island, Connecticut.....	B-16
Figure B-22. Quarterly and annual average toluene concentrations at Botanical Gardens, New York.....	B-16
Figure B-23. Quarterly and annual average toluene concentrations at Queens Community College, New York	B-17
Figure B-24. Quarterly and annual average toluene concentrations at East Lycoming, Pennsylvania.....	B-18
Figure B-25. Quarterly and annual average toluene concentrations at Lums Pond, Delaware	B-19
Figure B-26. Comparison of average hourly toluene concentrations at the Sherwood Island, Connecticut PAMS	B-20
Figure B-27. Comparison of average hourly toluene concentrations at the Botanical Gardens, New York PAMS	B-20
Figure B-28. Comparison of average hourly toluene concentrations at the Queens Community College, New York PAMS	B-21
Figure B-29. Comparison of average hourly toluene concentrations at the East Lycoming, Pennsylvania PAMS	B-22
Figure B-30. Comparison of average hourly toluene concentrations at the Lums Pond, Delaware PAMS	B-22
Figure B-31. Quarterly and annual average ethylbenzene concentrations at Sherwood Island, Connecticut	B-23
Figure B-32. Quarterly and annual average ethylbenzene concentrations at Botanical Gardens, New York	B-24
Figure B-33. Quarterly and annual average ethylbenzene concentrations at Queens Community College, New York	B-25
Figure B-34. Quarterly and annual average ethylbenzene concentrations at East Lycoming, Pennsylvania.....	B-26
Figure B-35. Quarterly and annual average ethylbenzene concentrations at Lums Pond, Delaware	B-26
Figure B-36. Comparison of average hourly ethylbenzene concentrations at the Sherwood Island, Connecticut PAMS	B-27
Figure B-37. Comparison of average hourly ethylbenzene concentrations at the Botanical Gardens, New York PAMS	B-27
Figure B-38. Comparison of average hourly ethylbenzene concentrations at the Queens Community College, New York PAMS	B-28
Figure B-39. Comparison of average hourly ethylbenzene concentrations at the East Lycoming, Pennsylvania PAMS	B-28
Figure B-40. Comparison of average hourly ethylbenzene concentrations at the Lums Pond, Delaware PAMS	B-29
Figure B-41. Quarterly and annual average formaldehyde concentrations at Botanical Gardens, New York	B-30
Figure B-42. Quarterly and annual average formaldehyde concentrations at Queens Community College, New York	B-31
Figure B-43. Quarterly and annual average formaldehyde concentrations at East Lycoming, Pennsylvania.....	B-31

Figure B-44. Quarterly and annual average formaldehyde concentrations at Lums Pond, Delaware	B-32
Figure B-45. Comparison of average hourly formaldehyde concentrations at the Botanical Gardens, New York PAMS	B-32
Figure B-46. Comparison of average hourly formaldehyde concentrations at the Queens Community College, New York PAMS	B-33
Figure B-47. Comparison of average hourly formaldehyde concentrations at the East Lycoming, Pennsylvania PAMS	B-33
Figure B-48. Quarterly and annual average acetaldehyde concentrations at Botanical Gardens, New York	B-34
Figure B-49. Quarterly and annual average acetaldehyde concentrations at Queens Community College, New York	B-35
Figure B-50. Quarterly and annual average acetaldehyde concentrations at East Lycoming, Pennsylvania	B-36
Figure B-51. Quarterly and annual average acetaldehyde concentrations at Lums Pond, Delaware	B-37
Figure B-52. Comparison of average hourly acetaldehyde concentrations at the Botanical Gardens, New York PAMS	B-37
Figure B-53. Comparison of average hourly acetaldehyde concentrations at the Queens Community College, New York PAMS	B-38
Figure B-54. Comparison of average hourly acetaldehyde concentrations at the East Lycoming, Pennsylvania PAMS	B-38

TABLES

Table ES-1. Top five statewide contributors to cancer risk, respiratory hazard, and neurological hazard according to NATA 2005 and NATA 2002	xviii
Table ES-2. Domain-wide summary of model performance evaluation results against 2002 NJ Air Toxics data	xxii
Table 1-1. List of urban air toxics.....	1-2
Table 1-2. Summary of model options and inputs selected for MANE-VU modeling.....	1-6
Table 2-1. Top five statewide contributors to cancer risk, respiratory hazard, and neurological hazard according to NATA 2005 and NATA 2002	2-1
Table 2-2. Top five contributors to cancer risk by county.....	2-2
Table 2-3. Top five contributors to respiratory health quotient by county	2-3
Table 2-4. Top five contributors to neurological health quotient by county	2-5
Table 3-1. Relevant parameters measured for the PAMS program	3-2
Table 3-2. Data files obtained from NJDEP	3-5
Table 3-3. Data obtained from the AQS Data Mart.....	3-6
Table 3-4. Summary of domestic carbon tetrachloride emissions in 2005.....	3-50
Table 4-1. Mapping CB05 species to CB4 species.....	4-7
Table 4-2. Domain-wide summary of model performance evaluation results against 2002 NJ Air Toxics data	4-11

Table 4-3. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for January.....	4-12
Table 4-4. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for February.....	4-12
Table 4-5. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for March.....	4-12
Table 4-6. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for April.....	4-13
Table 4-7. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for May	4-13
Table 4-8. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for June	4-13
Table 4-9. Domain-wide summary of regional haze model performance evaluation results against 2002 PAMS data for June.....	4-13
Table 4-10. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for July.....	4-14
Table 4-11. Domain-wide summary of regional haze model performance evaluation results against 2002 PAMS data for July	4-14
Table 4-12. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for August.....	4-14
Table 4-13. Domain-wide summary of regional haze model performance evaluation results against 2002 PAMS data for August	4-14
Table 4-14. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for September	4-15
Table 4-15. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for October	4-15
Table 4-16. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for November	4-15
Table 4-17. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for December.....	4-15
Table 4-18. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for January at Camden Lab	4-16
Table 4-19. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for January at Rutgers University.....	4-16
Table 4-20. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for January at Chester.....	4-16
Table 4-21. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for January at Elizabeth.....	4-16
Table 4-22. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for July at Camden Lab	4-17
Table 4-23. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for July at Rutgers University	4-17
Table 4-24. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for July at Chester.....	4-17
Table 4-25. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for July at Elizabeth.....	4-17

Table 4-26. Domain-wide summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for January.....	4-45
Table 4-27. Domain-wide summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for July	4-45
Table 4-28. Domain-wide summary of air toxics model performance evaluation results against 2002 PAMS data for July	4-45
Table 4-29. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for January at Camden Lab.....	4-46
Table 4-30. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for January at Rutgers University	4-46
Table 4-31. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for January at Chester	4-46
Table 4-32. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for January at Elizabeth	4-46
Table 4-33. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for July at Camden Lab.....	4-47
Table 4-34. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for July at Rutgers University.....	4-47
Table 4-35. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for July at Chester	4-47
Table 4-36. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for July at Elizabeth	4-47

Executive Summary

Air toxics are known or suspected to cause cancer or other serious health effects. Despite their ubiquity and role in increasing the risks of adverse health impacts, relatively little is known about the ambient levels of air toxics and contribution to health risks. Characterizing air toxics levels remains one of the key challenges to effectively controlling their risks.

The Northeast States for Coordinated Air Use Management (NESCAUM) and the New Jersey Department of Environmental Protection (NJDEP) completed a project to better characterize air toxics levels and to evaluate air quality models for use in assessing policies to reduce levels of air toxics. The purpose of this project is to provide a comprehensive assessment of priority air toxics in New Jersey based on three primary tasks: (1) analyze ambient air toxics data in New Jersey; (2) compare between existing ambient data, air quality modeling, and emission inventories; and (3) evaluate an updated model for use with air toxics. This report describes the activities performed to complete these tasks in order to characterize New Jersey air toxics and to assess the effectiveness of using existing model results for developing strategies that mitigate risks from air toxics.

ES-1. Top air toxics risk contributors in New Jersey

The U.S. Environmental Protection Agency's (US EPA's) National-Scale Air Toxics Assessment (NATA) is an ongoing US EPA study of air toxics and their associated cancer and non-cancer health risks at the census tract level across the United States. NESCAUM examined results from NATA 2002 and NATA 2005 to determine which air toxics posed the greatest risks to public health in New Jersey. The 2005 assessment includes 178 air toxics and was developed using 2005 emissions data with dispersion and photochemical modeling to estimate ambient concentrations and exposure levels. Ambient concentrations from major point sources, mobile sources, area sources, background concentrations, and formation in the atmosphere (secondary pollutants) were incorporated in the assessment.

The top five contributors to New Jersey cancer risk, respiratory hazard, and neurological hazard (statewide and by county) were extracted from NATA 2002 and NATA 2005 and are presented in Figure ES-1. Though the ordering of compounds differs, there is considerable overlap between the lists for 2002 and 2005 for each type of risk; there are no compounds in the top five list from NATA 2005 that are not also on the NATA 2002 list. At a statewide level, the top five contributors to cancer risk, respiratory hazard, and neurological hazard account for 77 percent, 97 percent, and 94 percent of total risk, respectively, according to NATA 2005.

Species that are in large part directly emitted by mobile sources play a major role in the cancer risk and respiratory health quotients. Benzene, acetaldehyde, 1,3-butadiene, acrolein, formaldehyde, and diesel engine emissions are mobile source air toxics (MSATs), and they represent four of the top five species for HAP-related cancer-risk and all of the top five for statewide respiratory hazard according to NATA 2005. The ubiquity of mobile sources and their significant contributions to statewide cancer and

Table ES-1. Top five statewide contributors to cancer risk, respiratory hazard, and neurological hazard according to NATA 2005 and NATA 2002

Rank	Top Five Compounds Contributing to NJ Cancer Risk	Top Five Compounds Contributing to NJ Respiratory Hazard	Top Five Compounds Contributing to NJ Neurological Hazard
# 1	Formaldehyde (2005) Benzene (2002)	Acrolein (2005 and 2002)	Cyanide compounds (2005) Xylenes (2002)
# 2	Benzene (2005) Carbon tetrachloride (2002)	Formaldehyde (2005) Acetaldehyde (2002)	Xylenes (2005) Cyanide compounds (2002)
# 3	Naphthalene (2005) Acetaldehyde (2002)	Acetaldehyde (2005) Formaldehyde (2002)	Manganese compounds (2005) Lead compounds (2002)
# 4	Acetaldehyde (2005) Naphthalene (2002)	Diesel engine emissions (2005 and 2002)	Methyl chloride (2005 and 2002)
# 5	Carbon tetrachloride (2005) 1,3-Butadiene (2002)	Naphthalene (2005) Methyl bromide (2002)	Lead compounds (2005) Manganese compounds (2002)

Source: NATA 2002 and 2005, US EPA.

non-cancer health hazard risk makes characterizing the risks from mobile source air toxics a priority.

It is important to be aware that NJDEP interprets the data from NATA differently than does the US EPA, because NJDEP uses different health benchmarks. Of special note, while the US EPA did not assess cancer risk from diesel exhaust in the 2005 NATA assessment, NJDEP uses the California cancer risk factor to determine the health benchmark for diesel particulate matter. Under the NJDEP approach, diesel exhaust is the highest contributor for cancer risk in the state, and would have been the greatest contributor to cancer risk in the 2005 NATA assessment had it been included. California has identified particulate matter in diesel exhaust (“soot”) as an air toxic due to its potential to cause cancer, premature death, and other health problems. In addition to particulate matter, diesel exhaust includes over 40 other cancer-causing substances. In New Jersey, the largest sources of diesel emissions are on-road and off-road vehicles, which include passenger cars, trucks, buses, construction equipment, agricultural equipment, locomotives, and marine vessels. Of additional concern in New Jersey is the presence of hundreds of small stationary diesel engines used for emergency back-up and high demand day electricity generation. With these myriad and widely distributed diesel emission sources, NJDEP estimates that the New Jersey county-level average cancer risk from exposure to diesel exhaust ranges from 39 to 916 in a million, with a statewide average of 324 in a million. The statewide diesel exhaust cancer risk is an order of

magnitude higher than that of formaldehyde, the highest ranked compound from NATA 2005.

In the NATA assessment, cancer risks in New Jersey are dominated by secondary formation (chemical production via reaction in the atmosphere) of formaldehyde, and to a lesser extent, of acetaldehyde, which together account for 37 percent of total cancer risk from air toxics statewide. Background accounts for 22 percent of total cancer risk from air toxics statewide, while on-road sources account for 19 percent, area sources for 14 percent, and non-road sources for 7 percent; major point sources account for less than 2 percent.

For respiratory hazard across the state, secondary formation is a dominant source, with 41 percent of respiratory hazard from ambient air toxics. Secondary production of acrolein alone accounts for 31 percent of respiratory risk from air toxics statewide. Area sources (24 percent), on-road sources (22 percent), and non-road sources (11 percent) make up the bulk of the remaining respiratory hazard from air toxics, while major point sources and background sources are both negligible (less than one percent) contributors.

Neurological hazard is dominated by area sources, which account for 45 percent of total statewide neurological hazard from air toxics; area sources of cyanide compounds alone account for 28 percent of statewide neurological hazard from air toxics. Other significant sources of neurological hazard are background (28 percent), point sources (10 percent), an on- (8 percent) and off-road (7 percent) sources.

ES-2. Air toxics monitoring

NESCAUM analyzed ambient monitoring data for individual air toxics species in New Jersey and surrounding states to determine the levels for air toxics in and around New Jersey. The data also provide a basis for identifying geographic and temporal patterns of air toxics levels. Prior to this report, analysis of the air toxics data collected by New Jersey's air toxics network had generally been limited to basic summary statistics on annual averages and inter-site comparisons. Additional analyses on temporal and spatial variability, characterization of localized impacts, and analysis of nearby sources will give a better understanding of the nature of air toxics in the region and allow better comparison to modeling data, thus aiding the development of control strategies. Figure ES-1 shows the locations of New Jersey monitors, which encompasses NJ Air Toxics monitoring sites and Photochemical Assessment Monitoring Stations (PAMS).

NESCAUM conducted the following analyses using the monitoring data: analysis of spatial characteristics including inter-site comparisons and analysis of local emission sources or transport patterns; analysis of temporal characteristics including descriptions of annual averages and seasonal variation; and examination of diurnal profiles when PAMS data were available.

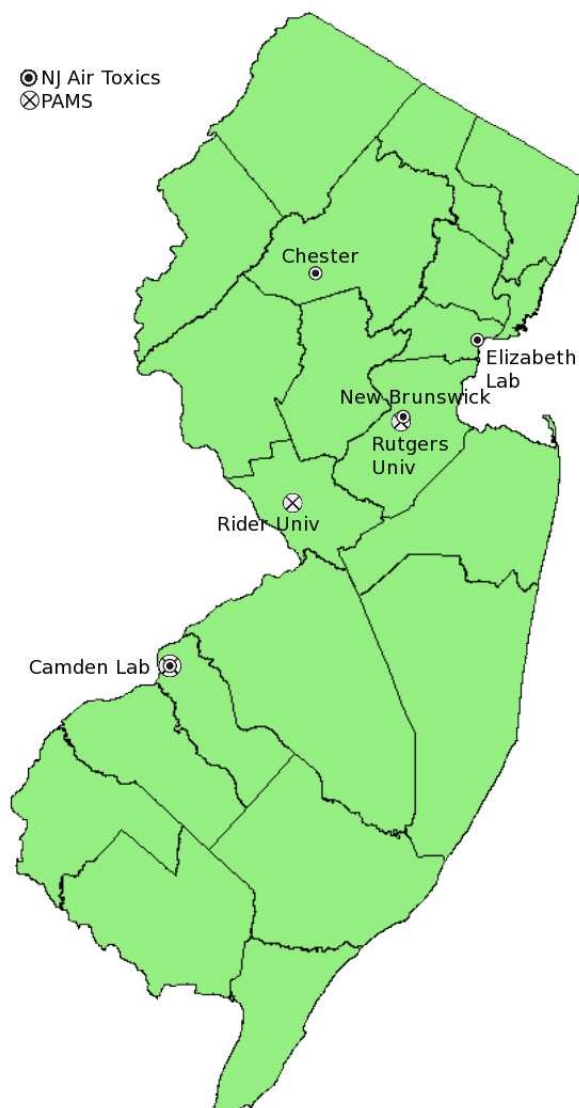
Monitoring results show that for species that are degraded but not produced by photochemistry (e.g., benzene, 1,3-butadiene, xylenes, toluene, ethylbenzene), there was an overall downward trend in annual average concentrations at most sites. For some species at some sites, however, the data did not clearly point toward a trend. The Elizabeth site typically had the highest concentrations of these species, followed by Camden, then New Brunswick, and Chester. Seasonal patterns for these compounds

were not uniform. For benzene and 1,3-butadiene, the summer concentrations often exceeded winter concentrations of the same year. For xylenes, toluene, and ethylbenzene, the relationship between seasonal concentrations was more variable. For diurnal trends, these species show early morning peaks, minimums in the early afternoon, and rising concentrations starting in the early evening.

For species with long atmospheric lifetimes and few known local sources, such as carbon tetrachloride, there is a rough upwards trend from year to year. Despite the lack of strong domestic emission sources, the upward trend is not surprising given the species' long lifetimes (decades). Because there are few large emission sources of carbon tetrachloride in and around New Jersey, monitors in the region are influenced by regional, national, and international sources, and therefore all sites had similar concentration levels. Summer concentrations often surpassed winter concentrations for a given year.

For species that are involved in and produced by photochemistry, in addition to anthropogenic emissions, concentrations rose steadily in some cases or were more variable from year to year. Concentrations between sites were also variable, and sites with the highest concentrations often changed from year to year. Summer concentrations for air toxics involved in photochemistry often surpassed winter concentrations. Because these compounds are formed in the atmosphere through chemical interaction requiring sunlight and relatively higher temperatures, it is expected that summer concentrations would be higher than those seen in the winter. Because NJDEP staff indicated that there are data quality issues related to the monitoring data for these species (Pietarinen, 2011), NESCAUM does not have a high degree of confidence in drawing conclusions about year-to-year trends or spatial relationships between the sites for these species. The PAMS data show that concentrations rise from early morning to an early-afternoon peak, and then gradually decline overnight until fresh emissions are introduced the next morning.

Figure ES-1. Air toxics monitoring sites in New Jersey



ES-3. Air quality modeling and model evaluation

While ambient measurements of air toxics can provide useful information on air toxics levels in the atmosphere and potential community impacts, air quality monitoring is subject to a number of constraints. Monitoring requires a significant investment of resources and, as a result, is often limited in spatial and temporal coverage. Air quality modeling can be used to supplement monitoring data for community-scale air toxics assessments. Before they can be used with confidence in such applications, however, air quality models must be evaluated and their limitations addressed to evaluate their ability to reproduce existing conditions and to determine their efficacy for testing potential policies and scenarios.

**Table ES-2. Domain-wide summary of model performance evaluation results
against 2002 NJ Air Toxics data**

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
January	ALD2	NJ_haze_2002	18	0.862	1.269	47.25	59.48	36.0	46.5
January	ALD2	NJ_tox_2002	18	0.862	1.670	93.76	93.76	61.3	61.3
January	FORM	NJ_haze_2002	18	1.798	0.934	-48.06	48.25	-58.3	58.6
January	FORM	NJ_tox_2002	18	1.798	1.399	-22.19	39.34	-22.9	42.7
January	OLE	NJ_haze_2002	18	1.577	1.822	15.56	93.95	70.1	89.2
January	OLE	NJ_tox_2002	18	1.577	2.610	65.56	130.90	94.9	107.9
January	PAR	NJ_haze_2002	18	8.284	71.863	767.44	767.44	158.7	158.7
January	PAR	NJ_tox_2002	18	8.284	96.384	1063.44	1063.44	167.8	167.8
January	TOL	NJ_haze_2002	18	2.463	2.581	4.80	85.93	36.6	72.4
January	TOL	NJ_tox_2002	18	2.463	3.069	24.60	105.89	51.0	85.7
January	XYL	NJ_haze_2002	18	1.306	4.425	238.78	238.78	108.8	108.8
January	XYL	NJ_tox_2002	18	1.306	1.935	48.15	83.40	38.0	63.9
July	ALD2	NJ_haze_2002	16	1.516	3.054	101.48	105.43	72.2	75.1
July	ALD2	NJ_tox_2002	13	1.595	3.772	136.53	136.53	83.5	83.5
July	FORM	NJ_haze_2002	16	4.511	4.434	-1.72	43.23	11.5	46.6
July	FORM	NJ_tox_2002	13	4.783	3.533	-26.14	40.79	-15.9	46.7
July	OLE	NJ_haze_2002	16	2.232	2.198	-1.53	85.98	50.0	80.1
July	OLE	NJ_tox_2002	13	2.388	1.751	-26.68	77.07	30.6	70.2
July	PAR	NJ_haze_2002	16	7.504	84.397	1024.64	1024.64	164.7	164.7
July	PAR	NJ_tox_2002	13	8.032	92.925	1057.01	1057.01	165.7	165.7
July	TOL	NJ_haze_2002	16	1.377	2.314	68.02	110.00	47.4	74.8
July	TOL	NJ_tox_2002	13	1.552	1.808	16.53	77.63	9.9	62.0
July	XYL	NJ_haze_2002	16	0.946	1.457	54.06	103.25	37.9	75.0
July	XYL	NJ_tox_2002	13	1.079	1.242	15.05	77.07	8.4	66.5

NJ_haze_2002 = regional haze model with CB4 chemistry; NJ_tox_2002 = NJ air toxics model with CB05 chemistry. NMB = Normalized mean bias. NME = Normalized mean error. MFB = Mean fractional bias. MFE = Mean fractional error.

Note: Slight differences in observational data values are the result of differences in model settings for start- and end-time. NJ_haze_2002 was set with 0:00 GMT as the start/end time; NJ_tox_2002 was set with 0:00 eastern as the start/end time.

The Community Multi-scale Air Quality Modeling System (CMAQ) is a state-of-the-science “one-atmosphere” system that treats major atmospheric and land processes and a range of species in a comprehensive framework. For the present analysis, NESCAUM built upon previous modeling work for regional haze using an older chemical mechanism (CB4 chemistry) to perform a 2002 simulation with a more recent version of CMAQ, version 4.7.1, that includes a new chemical mechanism capturing the chemistry of key air toxics species, “cb05txhg.”

Results of the model performance evaluations using “CB4 species” labels for the compared air toxics show similar performance for the older regional haze modeling and the present NJ air toxics modeling, as shown in Table ES-2. Though there are differences in individual aspects of their performance, the regional haze and air toxics models showed similar behavior. Both were broadly capable of reproducing temporal trends seen statewide, and both had common deficiencies (e.g., the gross overprediction of species with the paraffin carbon-carbon bond).

According to the summary-level statistics, model performance for air toxics is generally poor, perhaps due to poor representation of model chemistry or emissions in the model, with metrics varying significantly by month, species, and model. With a few exceptions, these results are generally biased high for both models, and the regional haze model shows somewhat lower levels of NMB (normalized mean bias) and NME (normalized mean error), but slightly higher levels of MFB (mean fractional bias) and MFE (mean fractional error).

Both models grossly overpredict paraffin bond volatile organic compounds (VOCs) by a factor of 10 or more in the summer; the regional haze model outperforms the air toxics model in the winter for paraffins, though the error is still seven-fold. Both models do better in their average monthly predictions for olefins, which are highly reactive. The regional haze model predictions for olefins are more accurate in the winter, with both models moderately overpredicting, while the air toxics model has lower error in the summer, when both models moderately underpredict. Monthly average predictions of formaldehyde are generally biased low within a factor of two and both models have reasonable performance. Similarly, formaldehyde is relatively well-predicted in both models, though better in the regional haze model. Higher aldehydes are biased high in both models in both summer and winter, though the regional haze model does slightly better, especially in winter. Monthly average predictions for higher aldehydes are generally within a factor of two, except for NMB in the summer, when predictions of both models are slightly higher. Predictions of toluene are biased slightly higher in both models and in both summer and winter, but generally match well against observations, with the regional haze model performing better in the winter and the air toxics model better in the summer. Both models performed reasonably well for xylenes, except for the regional haze model in the winter, which showed a four-fold overprediction. Summer monthly-average predictions of xylenes were within 15 percent for the air toxics model.

Both models failed to adequately reproduce levels of pollutants observed in New Jersey. These problems in model performance point primarily to the accuracy of the emissions inventory, because the models were largely able to reproduce other processes and patterns seen in the monitoring data. Additional analysis is required to confirm this result. Given the relative strengths and weaknesses identified here, we believe that these models are not adequate for assessing absolute levels of air toxics in New Jersey at this time. They may, however, be useful in providing insight about relative changes expected from policy or emission source measures.

1. INTRODUCTION

Regulated under the Clean Air Act, hazardous air pollutants (HAPs), or air toxics, are known or suspected to cause cancer or other serious health effects. Air toxics are emitted by a broad range of sources—from large and small facilities, to on- and off-road vehicles, to natural sources like wildfires and plant transpiration. Levels of air toxics and the risks posed by them vary by species, location, and time. Despite their ubiquity and role in increasing the risks of adverse health impacts, relatively little is known about the ambient levels of air toxics that contribute to health risks. Characterizing air toxics levels remains one of the key challenges to effectively controlling their risks.

Ambient measurements of air toxics can provide useful information on HAP levels in the atmosphere and potential community impacts. However, monitoring requires a significant investment of resources, can have limited precision for individual species, and is often limited in spatial and temporal coverage. As a result, air quality modeling has the potential to play a valuable role in community scale air toxics assessments. These models must be validated and their limitations addressed before they can be used with confidence in such applications.

The Northeast States for Coordinated Air Use Management (NESCAUM) and the New Jersey Department of Environmental Protection (NJDEP) completed a project to better characterize air toxics levels and to evaluate air quality models for use in assessing policies to reduce levels of air toxics. The purpose of this project is to provide a comprehensive assessment of priority air toxics in New Jersey based on three primary tasks: (1) analyze ambient air toxics data in New Jersey; (2) compare between existing ambient data, air quality modeling, and emission inventories; and (3) evaluate an updated model for use with air toxics. This report describes the activities performed to complete these tasks and presents the results.

1.1. Overview of air toxics

The 1990 Clean Air Act Amendments (CAAA, or “the Act”) identified 174 types of facilities or source categories that emit one or more of 188 toxic air pollutants listed under the Act. The CAAA requires that the U.S. Environmental Protection Agency (US EPA) to regulate emissions of air toxics by establishing maximum achievable control technology (MACT) standards for a set of large industrial sources, generally available control technologies (GACT) or MACT for smaller area sources, and technology and fuel standards for vehicles.

The US EPA has specified 33 air toxics from the larger list as urban air toxics, which pose the greatest risks to human health and the environment in urban areas. This list is presented in Table 1-1. On a national scale, six compounds pose the greatest risk to public health, including the air toxics benzene, formaldehyde, 1,3-butadiene, hexavalent chromium (chromium VI), and acrolein (Hafner et al., 2004). Sources of these compounds include mobile sources (benzene, formaldehyde, 1,3-butadiene), industrial sources and power plants (chromium VI and acrolein), and formation through chemical reactions in the atmosphere (formaldehyde and acrolein). Furthermore, while the US EPA does not list diesel particulate matter (PM) as a HAP, it is likely to pose the highest

public health risk from on-highway motor vehicle emission (Hafner et al., 2004). Although these compounds have been identified as posing a risk to human health and welfare, there remain only a limited number of federal standards and guidelines established as reference points for the evaluation of these risks. The lack of information on ambient concentrations of HAPs across the country has hindered efforts to assess potential health effects and policy initiatives to reduce ambient levels.

Recognizing the need for further action on air toxics, the US EPA's Strategic Plan, Sub-Objective 1.1.2 has set a goal to work with partner organizations to reduce air toxics' risk to public health by 2011. To reach this goal, the agency has set two targets, 19 percent reductions of toxicity weighted emission of air toxics in 2010 from the baseline year of 1993 for cancer risk and 55 percent reductions for non-cancer risk (US EPA, 2006). Understanding the nature and levels of air toxics in communities and developing reliable methods for predicting levels on a neighborhood scale is a necessary first step towards meeting these targets.

1.2. Air toxics in New Jersey

New Jersey has been tracking levels of air toxics since 1989. Data from monitors, and from air toxics risk models, have been used to assess the relative levels of risk in New Jersey. The US EPA's National-Scale Air Toxics Assessment (NATA) model predicts cancer and non-cancer risk levels from air toxics at the census tract level using predicted annual average air toxics levels. Monitoring data can be used to evaluate NATA (and other model) results, and also to characterize spatial and temporal variations of individual air toxics.

Examination of NATA reveals that a few air toxics are responsible for a large majority of key measures of risk relative to all air toxics. Among the compounds on this list are many air toxics emitted by mobile sources (e.g., benzene, acetaldehyde, acrolein, formaldehyde, diesel engine exhaust). It is important to know which compounds are the highest contributors to risk in New Jersey, because, according to NATA, the top five air toxics account for 77 percent of the cancer risk, 97 percent of the respiratory hazard, and

Table 1-1. List of urban air toxics

Acetaldehyde	Coke oven emissions	Manganese compounds
Acrolein	Dioxin	Mercury compounds
Acrylonitrile	Ethylene dibromide	Methylene chloride
Arsenic compounds	Propylene dichloride	Nickel compounds
Benzene	1, 3-Dichloropropene	Polychlorinated biphenyls (PCBs)
Beryllium compounds	Ethylene dichloride	Polycyclic organic matter (POM)
1, 3-Butadiene	Ethylene oxide	Quinoline
Cadmium compounds	Formaldehyde	1, 1, 2, 2-Tetrachloroethane
Carbon tetrachloride	Hexachlorobenzene	Perchloroethylene
Chloroform	Hydrazine	Trichloroethylene
Chromium compounds	Lead compounds	Vinyl chloride

Note: The above 33 species are air toxics that the US EPA has identified as presenting the greatest threat to public health in urban areas across the country.

Source: US EPA, "List of the 33 Urban Air Toxics." <http://www.epa.gov/ttn/atw/urban/list33.html>. Updated: September 2, 2008. Retrieved: June 3, 2011.

94 percent of the neurological hazard from air toxics statewide. These top risk contributors and a summary of usage, emissions sources, atmospheric behavior, and potential health effects for each cancer and non-cancer risk contributor are presented in Section 2.

It is important to be aware that NJDEP uses a different approach than that of NATA in assessing health risks from exposure to air toxics. Therefore, while this study only uses information from NATA to assess health risks in New Jersey, the approach used by NJDEP can result in different risk estimates from exposure to air toxics than that derived from NATA. Most notably, NJDEP's approach ranks diesel exhaust as the highest contributor for cancer risk in the state. NATA, however, does not assess diesel exhaust for cancer risk (although it does assess diesel exhaust for non-cancer risks). In 1998, California identified particulate matter in diesel exhaust ("soot") as an air toxic due to its potential to cause cancer, premature death, and other health problems (CARB, 2011). In addition to particulate matter, diesel exhaust includes over 40 other cancer-causing substances. In New Jersey, the largest sources of diesel emissions are on-road and off-road vehicles, which include passenger cars, trucks, buses, construction equipment, agricultural equipment, locomotives, and marine vessels. Of additional concern in New Jersey is the presence of hundreds of small stationary diesel engines used for emergency back-up and high demand day electricity generation. With these myriad and widely distributed diesel emission sources, NJDEP estimates that the New Jersey county-level average cancer risk from exposure to diesel exhaust ranges from 39 to 916 in a million. The statewide average diesel exhaust cancer risk is 324 in a million, which is an order of magnitude higher than that of formaldehyde, the compound having the highest ranked cancer risk in NATA 2005. Therefore, exposure to diesel exhaust would have been the highest ranked cancer risk in NATA 2005 had it been included (NJDEP, 2011).

1.3. Previous receptor modeling analysis

Positive matrix factorization (PMF) is a method for receptor modeling to detect source signatures in observational data. A primary feature of PMF is the ability to analyze a dataset across many factors, and to individually assign weights to data points to account for detection levels, missing data, and measurement uncertainty (Hopke, 2000). PMF allows the analyst to assert constraints to approximate natural systems. PMF has been successfully applied to identify factors related to variation in particulate matter measurements (see Hopke, 2000).

Applied to air toxics, PMF can identify source type signatures associated with observed air toxics levels. A previous study (Hopke, 2002) analyzed sources based on the PMF methodology for two receptors in New Jersey and one in Connecticut to determine factors associated with observed volatile organic compounds (VOCs), some of which are air toxics. The study used highly time-resolved (1-hr) data from New Jersey monitoring stations at Rider University and New Brunswick (see Section 3.1.2 for descriptions of these monitors). Hopke (2002) used a crude assumption for error model in estimating data uncertainties.

At the Rider University monitoring station, Hopke identified six factors associated with observed species concentrations. These were:

- (1) accumulation/aged air mass with an abundance of ethane and propane;
- (2) biogenic sources;
- (3) evaporative emissions from motor vehicles;
- (4) motor vehicle (gasoline and diesel fuel) combustion emissions;
- (5) accumulation/aged air mass¹ with small amounts of combustion products; and
- (6) combustion of diesel fuel, and solvents.

Hopke indicated a low degree of confidence in these assignments because he had limited experience interpreting VOC data. The assignments appear reasonable, though factor 4 in the list includes higher concentrations of trimethylbenzenes and xylenes that are not typically emitted as primary combustion products. Also, because observed levels for factor 4 are low, this may represent a regional mobile and area source signature profile, whereas factors 1, 3, and 5 may sufficiently account for near source combustion emissions. Factor 6 appears to represent industrial stationary and area sources. Further disaggregation may be possible.

Similarly, at the New Brunswick receptor, Hopke identified 10 factor profiles (not listed here). These were disaggregated into different evaporative emissions subsets, industrial/combustion signatures, biogenic sources, motor vehicles, and background sources. It is unlikely that subset categories of evaporative emissions will be useful in identifying unique sources or source categories for control.

Given the magnitude of concentrations in the identified factor profiles at New Brunswick and Rider University, background, biogenic, evaporative and combustion emissions appear to be the largest drivers of observed VOC levels.

1.4. Air toxics monitoring

The air toxics considered here vary considerably in their atmospheric behavior. The temporal and spatial variability of different species may be significant (Spicer et al., 1996; Luecken et al., 2006). McCarthy et al. (2007) examined the temporal variability of a number of air toxics across the United States, and found some distinct seasonal and diurnal behaviors for different groups of air toxics. Species like 1,3-butadiene and benzene show a diurnal pattern heavily influenced by mobile source emissions having a “morning peak”. Formaldehyde shows a daytime peak influenced by photochemical production, which may account for the majority of ambient formaldehyde (Luecken et al., 2006; McCarthy et al., 2007). Formaldehyde is produced from the oxidation of aldehydes, alkenes, and alcohols in the atmosphere. Acetaldehyde shows a daytime and morning peak because it is heavily influenced by photochemical production in addition to mobile sources (McCarthy et al., 2007).

Air toxics measurements are typically in the form of 24-hour averages. While these cannot be used to determine diurnal behavior, Photochemical Assessment Monitoring Stations (PAMS) measure some air toxics of interest at a higher temporal

¹ “Aged air mass” is synonymous with background.

resolution (1-3 hour samples, depending on species and season), and these may be used in conjunction with 24-hour average air toxics measurements to examine diurnal variation (Sistla and Aleksic, 2007).

Ambient measurements of air toxics provide the best indication of whether toxics are prevalent in ambient air and, in turn, affecting communities. Reliable measurements can provide accurate information on near-field ambient levels. Monitoring, however, also requires significant human and resource investment, can have limited precision for individual species, and is often limited in spatial and temporal coverage.

Air toxics monitors are usually sited in urban areas. Observations are measured at a single point, but the concentration gradients around monitors may differ for each species and location. Regional sources can be the most significant contributors to concentrations of some air toxics observed at a monitor, while in other locations or for other species, local sources may dominate. Spicer et al. (1996) found that if averaging times are long enough to smooth out the temporal patterns, measurements made in one neighborhood of some cities may be representative of most of the urban area. Luecken et al. (2006), however, found in some cases a 2 to 3-fold difference in measurements from monitors within one model grid cell. In a recent study of VOC spatial variation in Camden, New Jersey, spatial distributions in the neighborhood that included the Camden air toxics monitor were relatively uniform compared to measurements at a nearby hot spot for a number of short measurement campaigns (Zhu et al., 2008).

1.5. Air quality modeling

Ambient measurements of air toxics can provide useful information on air toxics levels in the atmosphere and potential community impacts. Monitoring, however, requires a significant investment of resources, can have limited precision for individual species, and is often limited in spatial and temporal coverage. Conversely, air quality modeling requires much lower investment of resources and can estimate air toxics levels with broad spatial and temporal coverage. As a result, air quality modeling has the potential to play a valuable role in community scale air toxics assessments. These models, however, must be properly evaluated and their limitations addressed before they can be used with confidence in such applications.

In addition to analyzing existing air toxics monitoring data and toxics emissions inventories, we examined existing and new modeling data for 2002. Existing data are available from simulations performed using the Community Multi-scale Air Quality modeling system (CMAQ) version 4.5 for the Mid-Atlantic/Northeast Visibility Union (MANE-VU) regional haze modeling (see NESCAUM, 2006) for 2002, 2009, and 2018. For the MANE-VU modeling, 2002 represented a baseline year for evaluating and demonstrating model performance, while 2009 and 2018 were used to model haze control strategies. Because the existing modeling was performed with the older CB4 chemistry mechanism, NESCAUM modeled air quality for selected months in 2002 using CMAQ version 4.7 and incorporated updated CB05 chemistry with added air toxics.

Key model parameters included for the MANE-VU effort are presented in Table 1-2. As detailed by NESCAUM (2006), the modeling for MANE-VU made use of the carbon bond IV (CB4) chemical mechanism. The new CB05 chemical mechanism

(Yarwood et al., 2005) replaced the CB4 mechanism as of CMAQ version 4.7 (June 2010 release). CB05 explicitly models some VOCs, many of which are air toxics. Though CB4 has representation for formaldehyde, acetaldehyde, and isoprene, the chemical placeholder for acetaldehyde is actually a lumped species, and the formaldehyde species represents both primary and secondary product. A broader range of air toxics are explicitly represented in CB05 including formaldehyde (primary and secondary, separately), acetaldehyde (explicitly), isoprene, 1,3-butadiene, acrolein, toluene, and individual isomers of xylene (*m*-, *o*-, and *p*-xylene).

Table 1-2. Summary of model options and inputs selected for MANE-VU modeling

<p>AQ Model: CMAQ v4.4</p> <p>Domains: 36-km (145x102), 12-km (172x172)</p> <p>AQ Vertical Layers: 22</p> <p>Years: 2002, 2009, 2018</p> <p>Chemistry: CB4 (Gery et al., 1989)</p>
<p>Met model: MM5</p> <p>Met options: Blackadar BL, explicit cloud representation, KF cumulus parameterization</p> <p>Met model vertical layers: 29</p>
<p>Emissions: SMOKE v2.1</p> <p>Biogenics: BEIS3 w/ BELD3</p> <p>Onroad: MOBILE6 (RPO data)</p> <p>Other: CEM data</p> <p>Canada: 2000. Mexico: 1999</p>

Source: NESCAUM (2006).

1.6. Layout of this report

This report contains three separate and complementary analyses, which are described in the following sections:

- **Section 2** provides an overview of the US EPA National Air Toxics Assessment (NATA), which NESCAUM used to identify air toxics of highest concern.
- **Section 3** presents the results of ongoing air quality monitoring for air toxics across New Jersey, and discusses year-to-year trends as well as geographic, seasonal, and daily patterns observed in the monitoring data.
- **Section 4** presents the results of an air quality modeling simulation and performance evaluation, which is used to assess the ability of the model to reproduce monitored levels of air toxics in New Jersey.
- **Section 5** presents a summary of the findings from Sections 2 through 4.
- **Section 6** is the reference list.

2. ANALYSIS OF THE NATIONAL AIR TOXICS ASSESSMENT (NATA)

NESCAUM examined results from the US EPA's National-Scale Air Toxics Assessment (NATA) model to determine which air toxics posed the greatest risks to public health in New Jersey. The NATA model predicts cancer and non-cancer risk levels from air toxics at the census tract level using predicted annual average air toxics levels. NATA was developed as a screening tool to assist communities and regulatory bodies in assessing their air toxic priorities by identifying those air toxics that pose the greatest risk to the public. As part of NATA development, air toxics monitoring, modeling, emissions inventories, exposures, and health effects research are regularly updated. New assessments are projected to be performed with the release of updated air toxics emissions inventories that occur every three years. At the end of 2011, the US EPA had completed three air toxics assessments and was in the process of finalizing a fourth. A version of the fourth assessment, based upon the 2005 National Emissions Inventory (NEI), was released in February 2011. This version incorporates data from the US EPA's Motor Vehicle Emission Simulator (MOVES), an updated mobile source emissions model.

NJDEP interprets the data from NATA differently than does the US EPA, because NJDEP uses different health benchmarks for some compounds. Of special note, while the US EPA did not assess cancer risk from diesel exhaust, NJDEP uses the California

Table 2-1. Top five statewide contributors to cancer risk, respiratory hazard, and neurological hazard according to NATA 2005 and NATA 2002

Rank	Top Five Compounds Contributing to NJ Cancer Risk	Top Five Compounds Contributing to NJ Respiratory Hazard	Top Five Compounds Contributing to NJ Neurological Hazard
# 1	Formaldehyde (2005) Benzene (2002)	Acrolein (2005 and 2002)	Cyanide compounds (2005) Xylenes (2002)
# 2	Benzene (2005) Carbon tetrachloride (2002)	Formaldehyde (2005) Acetaldehyde (2002)	Xylenes (2005) Cyanide compounds (2002)
# 3	Naphthalene (2005) Acetaldehyde (2002)	Acetaldehyde (2005) Formaldehyde (2002)	Manganese compounds (2005) Lead compounds (2002)
# 4	Acetaldehyde (2005) Naphthalene (2002)	Diesel engine emissions (2005 and 2002)	Methyl chloride (2005 and 2002)
# 5	Carbon tetrachloride (2005) 1,3-Butadiene (2002)	Naphthalene (2005) Methyl bromide (2002)	Lead compounds (2005) Manganese compounds (2002)

Source: NATA 2002 and 2005, US EPA.

cancer risk factor to determine the health benchmark for diesel particulate matter. Under the NJDEP approach, diesel exhaust is the highest contributor for cancer risk in the state, with the next five compounds of highest risk in the same order as in the 2005 NATA top five. Additional information on how NJDEP interprets the NATA results is available on its website <http://www.nj.gov/dep/airtoxics/>. This site has maps of New Jersey benchmark risk estimates at the census tract level along with tabulated county and statewide averages. In this report, we present only the results from the US EPA NATA study.

2.1. About NATA 2005

The 2005 NATA assessment includes 178 air toxics and was developed using 2005 emissions data, dispersion and photochemical modeling to determine ambient concentrations and exposure, and non-cancer hazard reference concentrations. Ambient concentrations from major point sources, mobile sources, area sources, background concentrations, and formation in the atmosphere (secondary formation) were incorporated in the assessment. Background concentrations, which are not represented in the dispersion models and are meant to capture concentrations originating from long range transport, natural emissions, and other unidentified emissions sources, were estimated from ambient concentrations, comparison to areas with similar population levels, or

Table 2-2. Top five contributors to cancer risk by county

County	#1	#2	#3	#4	#5
Atlantic	Formaldehyde	Benzene	Carbon tetrachloride	Acetaldehyde	Naphthalene
Bergen	Formaldehyde	Benzene	Naphthalene	Acetaldehyde	1,3-butadiene
Burlington	Formaldehyde	Benzene	Acetaldehyde	Carbon tetrachloride	Naphthalene
Camden	Formaldehyde	Benzene	Acetaldehyde	Naphthalene	Carbon tetrachloride
Cape May	Formaldehyde	Benzene	Carbon tetrachloride	Acetaldehyde	1,3-butadiene
Cumberland	Formaldehyde	Benzene	Carbon tetrachloride	Acetaldehyde	Naphthalene
Essex	Formaldehyde	Benzene	Naphthalene	1,3-butadiene	Acetaldehyde
Gloucester	Formaldehyde	Benzene	Acetaldehyde	Carbon tetrachloride	Naphthalene
Hudson	Formaldehyde	Benzene	Naphthalene	Acetaldehyde	1,3-butadiene
Hunterdon	Formaldehyde	Benzene	Carbon tetrachloride	Acetaldehyde	Naphthalene
Mercer	Formaldehyde	Benzene	Acetaldehyde	Naphthalene	Carbon tetrachloride
Middlesex	Formaldehyde	Benzene	Naphthalene	Acetaldehyde	1,3-butadiene
Monmouth	Formaldehyde	Benzene	Carbon tetrachloride	Acetaldehyde	Naphthalene
Morris	Formaldehyde	Benzene	Acetaldehyde	Naphthalene	Carbon tetrachloride
Ocean	Formaldehyde	Benzene	Carbon tetrachloride	Acetaldehyde	Naphthalene
Passaic	Formaldehyde	Benzene	Naphthalene	Acetaldehyde	1,3-butadiene
Salem	Formaldehyde	Benzene	Carbon tetrachloride	Acetaldehyde	1,1,2-trichloroethane
Somerset	Formaldehyde	Benzene	Acetaldehyde	Carbon tetrachloride	Naphthalene
Sussex	Formaldehyde	Benzene	Carbon tetrachloride	Acetaldehyde	Naphthalene
Union	Formaldehyde	Benzene	Naphthalene	1,3-butadiene	Acetaldehyde
Warren	Formaldehyde	Benzene	Carbon tetrachloride	Acetaldehyde	Naphthalene

Source: NATA 2005, US EPA.

emissions data where possible (ICF International, 2011).

The NEI and MOVES emissions were processed for input into the dispersion models. In addition, secondary formation (produced in the atmosphere rather than directly emitted) of formaldehyde, acetaldehyde, acrolein, and 1,3-butadiene was estimated using the Community Multi-scale Air Quality (CMAQ) modeling system. The Assessment System for Population Exposure Nationwide (ASPEN) model was run for area and mobile source emissions, and the Human Exposure Model (HEM-3) was run for stationary source emissions. ASPEN and HEM-3 are based on data from the 2000 Census and provide ambient concentrations for each census tract or block. HAPEM5, a screening-level exposure model, can be used with the ASPEN, HEM-3, and CMAQ ambient concentration predictions to estimate a population-weighted median exposure concentration for each census tract. From this exposure concentration, cancer risk and non-cancer hazards can be estimated for each HAP with associated health data. HAPEM5 was not run for NATA 2005. Instead, as with NATA 2002, exposure concentrations were estimated by multiplying the 2005 modeled ambient concentrations by a ratio of the 1999 HAPEM exposure concentrations to 1999 ambient concentrations for the same species, census tract, and source type. These exposure concentrations were used with unit risk estimates and inhalation reference concentrations in order to estimate lifetime cancer risk and respiratory and neurological hazard quotients.

While air toxics come from a variety of emissions sources, mobile sources are believed to contribute a large percentage of the cancer risk from outdoor air toxics

Table 2-3. Top five contributors to respiratory health quotient by county

County	#1	#2	#3	#4	#5
Atlantic	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Methyl bromide
Bergen	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Naphthalene
Burlington	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Naphthalene
Camden	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Naphthalene
Cape May	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Methyl bromide
Cumberland	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Methyl bromide
Essex	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Naphthalene
Gloucester	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Chlorine
Hudson	Acrolein	Diesel engine emissions	Formaldehyde	Acetaldehyde	Methyl bromide
Hunterdon	Acrolein	Acetaldehyde	Formaldehyde	Diesel engine emissions	Naphthalene
Mercer	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Methyl bromide
Middlesex	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Naphthalene
Monmouth	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Naphthalene
Morris	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Naphthalene
Ocean	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Methyl bromide
Passaic	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Methyl bromide
Salem	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Naphthalene
Somerset	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Naphthalene
Sussex	Acrolein	Acetaldehyde	Formaldehyde	Diesel engine emissions	Naphthalene
Union	Acrolein	Formaldehyde	Acetaldehyde	Diesel engine emissions	Naphthalene
Warren	Acrolein	Acetaldehyde	Formaldehyde	Diesel engine emissions	Naphthalene

Source: NATA 2005, US EPA.

sources (US EPA, 1994). As with NATA 2002, NATA 2005 did not consider cancer risk from diesel exhaust emissions as for other air toxics because the US EPA felt there was not enough data to quantitatively estimate the carcinogenic potency of diesel particulate matter. Nevertheless, it is believed that diesel exhaust poses one of the largest relative risks to the population at large (US EPA, 2009a), and NJ DEP, using a cancer risk factor taken from California, finds diesel exhaust to have the highest cancer risk among air toxics in New Jersey (NJ DEP 2011). Diesel exhaust is a complex gas-particle phase mixture of both known and unknown compounds that include confirmed carcinogens like benzene (US EPA, 2002). Increased lung cancer risk has been linked to diesel exhaust in a number of epidemiology studies, and diesel PM contributes to non-cancer health risks as well. Fine particulate matter, like diesel PM, has been linked to respiratory and cardiovascular health effects. While the US EPA does not consider cancer risks, non-cancer hazards have been assessed for diesel exhaust in NATA 2005 (ICF International, 2011).

2.2. Results from NATA 2005

According to NATA 2005, the average cancer risk from air toxics in the United States is 0.000050 (50 in a million), compared to 0.000036 (36 in a million) as estimated by the previous assessment, NATA 2002. Cancer risks in NATA are presented as the lifetime probability of developing cancer as a result of exposure to air toxics over a 70-year lifetime. Because these risk values are probabilities, they are not anticipated cancer incidences.

The difference in reported cancer risk between 2002 and 2005 is mostly attributable to changes in the methods for estimating cancer risk and exposure for formaldehyde.² Per the US EPA, it is not meaningful to directly compare the results of NATA 2005 to those from NATA 2002 because of the changes in methodology (US EPA, 2011). NATA 2002, based on the 2002 NEI, was released in June 2009 and included 180 air toxics plus diesel PM, with cancer risks and non-cancer health hazards assessed for the 124 compounds for which chronic exposure health data existed. The assessment gives an estimate of the cancer risks a population might face if it was exposed to 2002 emission levels for an assumed 70 year lifetime. Non-cancer risks are presented as hazard quotients. A hazard quotient is a ratio between the estimated exposure concentration and a reference concentration below which adverse health effects are unlikely. NJDEP uses risk ratios against health benchmarks, rather than adopting the US EPA's terminology for air toxics risk. A health benchmark is the level below which no observable health effect is expected to result. A risk ratio is the actual level divided by the health benchmark. Therefore, a risk ratio below 1.0 indicates that no health effects are expected.

² NATA 2005 made use of a chemical transformation available in the CMAQ model to estimate secondary formation of formaldehyde (and other substances), which accounts for approximately 90 percent of ambient concentrations, and which was not accounted for in NATA 2002. Furthermore, NATA 2005 makes use of an estimate of cancer potency for formaldehyde that is approximately 2,200 times the value of the NATA 2002 estimate. These changes result in a cancer risk contribution of 22 per million for NATA 2005 compared to a less than 1 per million contribution for NATA 2002 (ICF International, 2011).

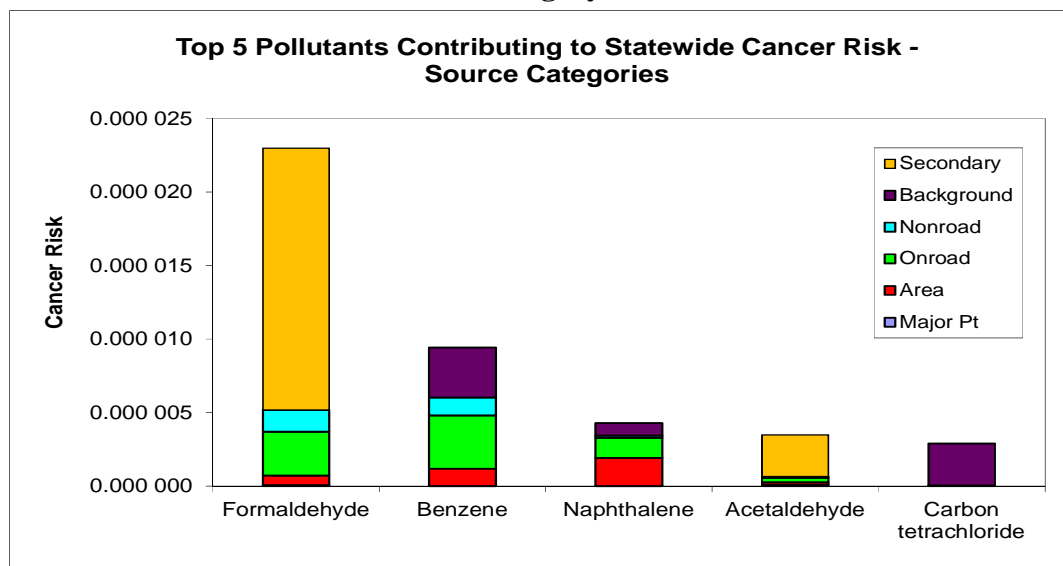
The top five contributors to New Jersey cancer risk, respiratory hazard, and neurological hazard (statewide and by county) were extracted from NATA 2002 and NATA 2005 and are presented in Table 2-1. Though the ordering of compounds differs, there is considerable overlap between the lists for 2002 and 2005 for each type of risk; there are no compounds in the top five list from NATA 2005 that are not also on the

Table 2-4. Top five contributors to neurological health quotient by county

County	#1	#2	#3	#4	#5
Atlantic	Methyl chloride	Cyanide compounds	Manganese compounds	Xylenes	Lead compounds
Bergen	Cyanide compounds	Xylenes	Manganese compounds	Methyl chloride	Lead compounds
Burlington	Manganese compounds	Cyanide compounds	Methyl chloride	Lead compounds	Xylenes
Camden	Cyanide compounds	Manganese compounds	Xylenes	Methyl chloride	Lead compounds
Cape May	Methyl chloride	Manganese compounds	Lead compounds	Cyanide compounds	Xylenes
Cumberland	Methyl chloride	Manganese compounds	Cyanide compounds	Lead compounds	Xylenes
Essex	Cyanide compounds	Xylenes	Manganese compounds	Methyl chloride	Lead compounds
Gloucester	Cyanide compounds	Methyl chloride	Manganese compounds	Xylenes	Lead compounds
Hudson	Cyanide compounds	Xylenes	Manganese compounds	Lead compounds	Methyl chloride
Hunterdon	Methyl chloride	Cyanide compounds	Manganese compounds	Lead compounds	Xylenes
Mercer	Cyanide compounds	Manganese compounds	Methyl chloride	Xylenes	Lead compounds
Middlesex	Xylenes	Cyanide compounds	Manganese compounds	Methyl chloride	Lead compounds
Monmouth	Cyanide compounds	Methyl chloride	Xylenes	Manganese compounds	Lead compounds
Morris	Cyanide compounds	Methyl chloride	Xylenes	Manganese compounds	Lead compounds
Ocean	Methyl chloride	Manganese compounds	Cyanide compounds	Xylenes	Lead compounds
Passaic	Cyanide compounds	Xylenes	Methyl chloride	Manganese compounds	Lead compounds
Salem	Methyl chloride	Manganese compounds	Cyanide compounds	Xylenes	Lead compounds
Somerset	Cyanide compounds	Methyl chloride	Xylenes	Manganese compounds	Lead compounds
Sussex	Methyl chloride	Cyanide compounds	Manganese compounds	Lead compounds	Xylenes
Union	Manganese compounds	Xylenes	Cyanide compounds	Lead compounds	Methyl chloride
Warren	Cyanide compounds	Methyl chloride	Manganese compounds	Lead compounds	Xylenes

Source: NATA 2005, US EPA.

Figure 2-1. Top five pollutants contributing to New Jersey cancer risk by source category



Note: Values presented in fractional cancer risk.

Source: NATA 2005

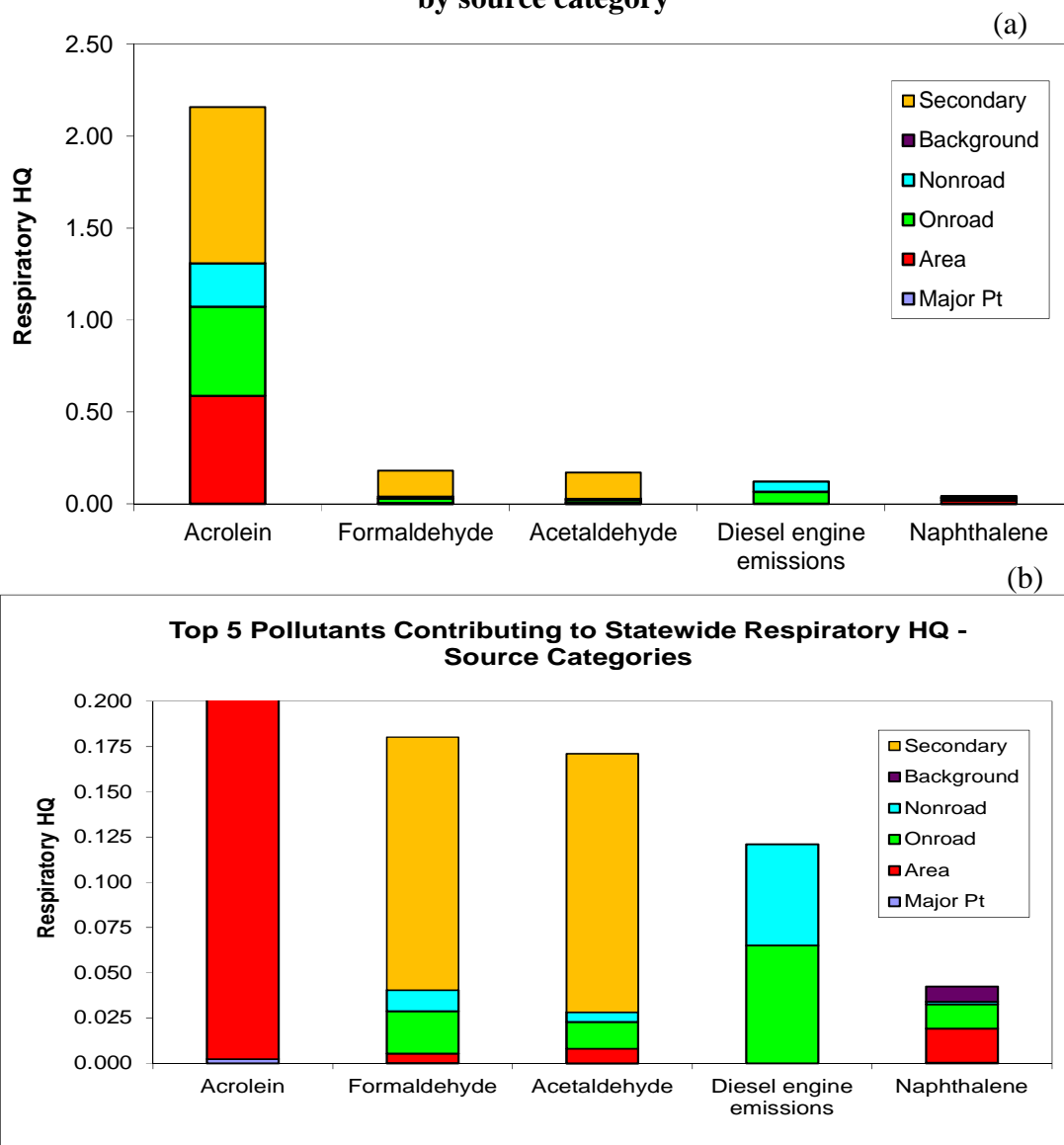
NATA 2002 list. At a statewide level, the top five contributors to cancer risk, respiratory hazard, and neurological hazard account for 77 percent, 97 percent, and 94 percent of total risk, respectively, according to NATA 2005. A summary of usage, emissions sources, atmospheric behavior, and potential health effects for each of the top five statewide cancer and non-cancer risk contributors is presented in Section 2.3.

Results at the county level for NATA 2005, as shown in Table 2-2 through Table 2-4, generally indicate agreement with the statewide results. For cancer risk, formaldehyde and benzene (in that order) are the top air toxics in each county in New Jersey. The third through fifth most important air toxics contributing to cancer risk in individual counties in New Jersey vary depending on the county, and include acetaldehyde, carbon tetrachloride, naphthalene, 1,3-butadiene, and 1,1,2-trichloroethane.

As seen in Table 2-3, the most important contributor to respiratory hazard in all New Jersey counties is acrolein; the next three highest are typically in the order formaldehyde, acetaldehyde, and diesel engine exhaust. In some counties, however, acetaldehyde is a more important contributor than formaldehyde, except in Hudson County where diesel engine exhaust is a more important contributor followed by formaldehyde and acetaldehyde. Other species among the top five contributors to respiratory hazard in individual counties are methyl bromide, naphthalene, and (in the case of Gloucester County) chlorine.

Table 2-4 shows the top five contributors by county to neurological health quotient. The top five contributors are the same across each county, but the order in which they occur varies from county to county. The top contributors are cyanide compounds, lead compounds, manganese compounds, methyl chloride, and xylenes. The aggregate neurologic risk ratio is well below the 1.0 threshold, indicating that expected neurologic risk from air toxics is of low importance in New Jersey.

Figure 2-2. Top five pollutants contributing to New Jersey respiratory hazard index by source category



Note: A hazard index is a sum of hazard quotients for substances that affect the same target organ. A hazard quotient is a ratio between the estimated exposure concentration and a reference concentration below which adverse health effects are unlikely. *Source:* NATA 2005

In this figure, (b) is a zoom on (a) to clearly show the emissions sources of pollutants two through five.

As shown in Figure 2-1 through Figure 2-3, species that are in large part directly emitted by mobile sources play a major role in the cancer risk and respiratory health quotients. Benzene, acetaldehyde, 1,3-butadiene, acrolein, formaldehyde, and diesel engine emissions are mobile source air toxics (MSATs), and they represent three of the top five statewide contributors to HAP-related cancer-risk and four of the top five species contributing to the statewide respiratory hazard from NATA 2002. For NATA 2005, they represent four of the top five species for HAP-related cancer-risk and all of the top five for statewide respiratory hazard. Diesel engine emissions are not included in either

NATA 2002 or 2005 estimates of cancer risk. Many epidemiological studies indicate that the complex gas-particle mixture emitted from diesel engines may contribute significantly, and may even be the most significant contributor, to human cancer risk from mobile sources (CARB, 2005; US EPA, 2002). NJDEP's approach, which uses a diesel cancer risk factor from California, ranks diesel exhaust particulate matter as the highest cancer risk among air toxics in New Jersey (NJ DEP 2011).

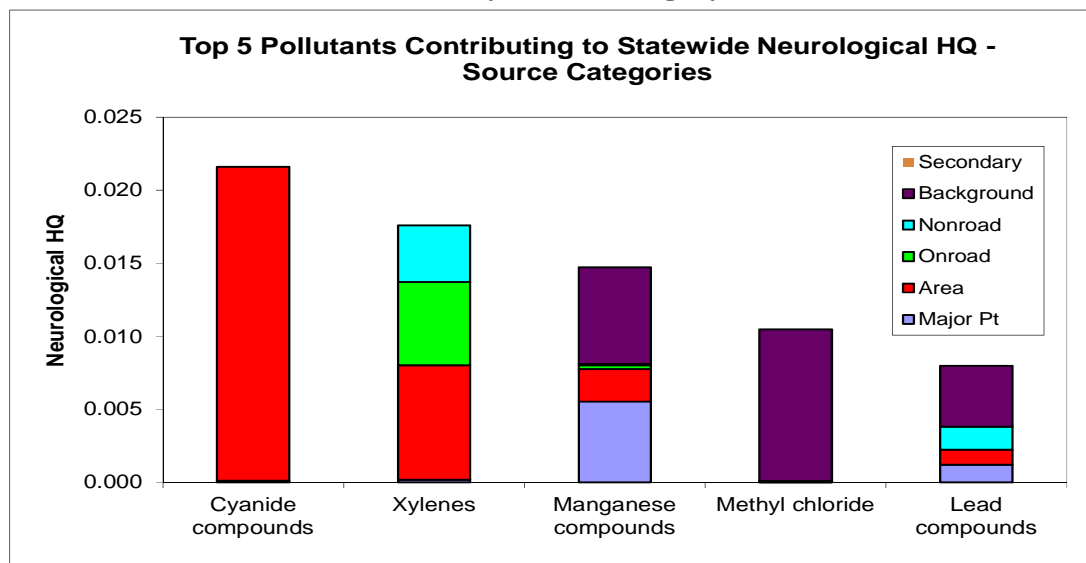
The ubiquity of mobile sources and their significant contributions to statewide cancer and non-cancer health hazard risk makes characterizing the risks from mobile source air toxics a priority. Because of the information obtained through evaluation of the NATA 2002 and 2005 results, NESCAUM focused its assessment of monitored air toxics on MSATs (see Section 3). While we will not address diesel exhaust separately, a number of the mobile source species that we will examine (e.g., formaldehyde, acetaldehyde, benzene, etc.) make up part of the complex mixture that is emitted by diesel engines (US EPA, 2002).

As seen in Figure 2-1, cancer risks in New Jersey derived from NATA are dominated by secondary formation (chemical production via reaction in the atmosphere) of formaldehyde, and to a lesser extent, of acetaldehyde, which together account for 37 percent of total cancer risk from air toxics statewide. Background accounts for 22 percent of total cancer risk from air toxics statewide, while on-road sources account for 19 percent, area sources for 14 percent, and non-road sources for 7 percent; major point sources account for less than 2 percent. As previously mentioned, NATA does not assess diesel exhaust for cancer risk, whereas NJDEP uses a cancer risk factor from California that ranks exposure to diesel exhaust particulate matter as the highest cancer risk in the state among the air toxics (NJDEP, 2011).

For respiratory hazard across the state, as shown in Figure 2-2, secondary formation is a dominant source, with 41 percent of respiratory hazard from ambient air toxics. Secondary production of acrolein alone accounts for 31 percent of respiratory risk from air toxics statewide. Area sources (24 percent), on-road sources (22 percent), and non-road sources (11 percent) make up the bulk of the remaining respiratory hazard from air toxics, while major point sources and background sources are both negligible contributors (less than 1 percent).

Neurological hazard is dominated by area sources, which account for 45 percent of total statewide neurological hazard from air toxics; area sources of cyanide compounds alone account for 28 percent of statewide neurological hazard from air toxics. Other sources of neurological hazard are background (28 percent), point sources (10 percent), and on-road (8 percent) and off-road (7 percent) sources. Figure 2-3 displays additional details of the top five neurological hazard air toxics and their sources.

Figure 2-3. Top five pollutants contributing to New Jersey neurological hazard index by source category



Note: A hazard index is a sum of hazard quotients for substances that affect the same target organ. A hazard quotient is a ratio between the estimated exposure concentration and a reference concentration below which adverse health effects are unlikely. *Source: NATA 2005*

2.3. Sources, atmospheric processes, and health effects of risk-driving compounds

Profiles of each of the top risk-contributors in New Jersey (the compounds listed in Table 2-1) from NATA 2002 and 2005 are presented here in alphabetical order by compound. These profiles include information on emission sources, atmospheric processes, and health effects. Toluene and ethylbenzene are also described in this section and assessed in this report, though these compounds are lower risk contributors according to the results from these NATA studies.

Acetaldehyde

Usage, emissions sources and atmospheric processes

Acetaldehyde is used as a solvent, as a preservative, and in the production of other chemicals. It is produced in the atmosphere, is a component of motor vehicle exhaust, and is also a product of incomplete wood combustion (US EPA, 2009b). Atmospheric acetaldehyde concentrations are influenced by direct emissions as well as chemical production and removal. Acetaldehyde is formed in the atmosphere when other organic compounds degrade, and they are removed by direct photolysis and reactions with the hydroxyl radical (Seinfeld and Pandis, 1998). Acetaldehyde concentrations tend to be higher in the summer months due to higher temperatures, higher photolysis rates, and higher biogenic emissions. Photochemical production accounts for more than half of the modeled total aldehyde concentrations but that the magnitude varies by location (Luecken et al., 2006). While direct emissions of aldehydes are important, especially in urban areas during the winter, due to the large role that photochemical production may

play in ambient aldehyde concentrations, VOC precursors must be considered in control strategies for these species.

Health impacts

Short-term exposure to acetaldehyde leads to irritation of the respiratory tract, skin, and eyes. Long-term exposure results in biological effects similar to those produced by alcoholism. The US EPA (2009b) has classified acetaldehyde as a probable human carcinogen (group B2).

Acrolein

Usage, emissions sources and atmospheric processes

Acrolein can be formed during the combustion of fuels or other organic matter. It is used as a biocide and as an intermediate in acrylic acid synthesis (US EPA, 2009b). Acrolein can be emitted directly or formed via photochemical production from 1,3-dienes (e.g., 1,3-butadiene). It is also destroyed in the atmosphere through reactions with the hydroxyl radical. Photochemical production is estimated to account for 30-50 percent of annual acrolein concentrations (Luecken et al., 2006). Photochemical production contributes slightly more to summer acrolein concentrations than winter.

Health impacts

Short-term exposure may lead to eye, nose and throat irritation. Chronic exposure of acrolein by inhalation may lead to irritation of the upper-respiratory tract and congestion (US EPA, 2009b).

Benzene

Usage, emissions sources and atmospheric processes

Benzene is emitted from mobile sources, gasoline stations, and coal and oil combustion, and is also used in industrial solvents (US EPA, 2009b). Benzene is also found in tobacco smoke. While not produced in the atmosphere, benzene undergoes chemical removal via reaction with the hydroxyl radical. The hydroxyl radical, which is produced photochemically, will be more prevalent during daylight hours and warmer months, and so benzene will be removed more efficiently via chemistry during the summer as opposed to the winter. Changes in emission patterns or boundary layer heights may also affect the seasonal concentrations of benzene (Luecken et al., 2006). Benzene has an estimated atmospheric lifetime of 12 days due to reaction with the hydroxyl radical (Seinfeld and Pandis, 1998). As a result, benzene concentrations can be influenced by local emissions or long-range transport from distant emissions sources.

Health impacts

Short-term exposure via inhalation may lead to dizziness, drowsiness, headaches, and irritation of the respiratory tract, eyes, and skin or, at very high levels, unconsciousness. Chronic exposure may lead to blood disorders, adverse reproductive effects, and

leukemia. The US EPA (2009b) has classified benzene as a known human carcinogen (group A).

1,3-Butadiene

Usage, emissions sources and atmospheric processes

1,3-Butadiene is emitted from mobile sources, industrial facilities (such as those involved in the production of rubber and plastics), forest fires, and, like many other compounds described here, can be found in tobacco smoke (US EPA, 2009b). Motor vehicle exhaust is the most pervasive source of 1,3 butadiene emissions. While widely emitted, 1,3-butadiene has a lifetime on the order of hours due to degradation in the atmosphere, and atmospheric concentrations tend to be low. In the atmosphere, 1,3-butadiene can be oxidized to form acrolein (Luecken et al., 2006).

Health impacts

Short-term exposure may result in irritation of the eyes, nose, throat, and lungs and, in cases of significant acute exposure, may also lead to blurred vision, fatigue, headache, and vertigo. Chronic exposure has been linked to cardiovascular disease and leukemia or other cancers. The US EPA (2009b) has classified 1,3-butadiene as a probable human carcinogen (group B2).

Carbon tetrachloride

Usage, emissions sources and atmospheric processes

Carbon tetrachloride was once widely used in the production of refrigerants, aerosol can propellants, in dry cleaning, as a chemical solvent, and as a pesticide/fumigant. Usage is now mainly limited to industrial applications (ATSDR, 2005; US EPA, 2009b). Carbon tetrachloride has a long atmospheric residence time of about 42 years (Seinfeld and Pandis, 1998). The main atmospheric sink is likely photochemical destruction in the stratosphere (Allen et al., 2009). Carbon tetrachloride is an ozone-depleting substance and greenhouse gas (Allen et al., 2009).

Health impacts

Short-term exposures via inhalation or oral intake may cause headache, weakness, nausea or vomiting. If the short-term exposure is high or if exposure is chronic, it may lead to liver and kidney damage. The US EPA (2009b) has classified carbon tetrachloride as a probable human carcinogen (group B2).

Cyanide compounds

Usage, emissions sources and atmospheric processes

Cyanide, which is emitted by biogenic and anthropogenic processes, may be produced naturally by certain types of bacteria, fungi, and algae and can be found in certain plants and foods (ATSDR, 2006; US EPA, 2009b). It is used in electroplating, metallurgy, as an intermediate in chemical production, and as an insecticide. Most cyanide in the

atmosphere comes from motor vehicle exhaust, industrial and chemical processing, and municipal waste incinerators (US EPA, 2009b). Cigarette smoke also contains cyanide (ATSDR, 2006). Most cyanide in the atmosphere is in the form of gaseous hydrogen cyanide with a small amount in fine particles. The half-life of cyanide is on the order of 1 to 3 years (ATSDR, 2006).

Health impacts

Cyanide is very toxic, and acute inhalation exposure to high amounts of hydrogen cyanide ($> 100 \text{ mg/m}^3$) will result in death. At lower levels, short-term exposure may result in headache, nausea, irritation of eyes and skin, and weakness. Chronic exposures may result in adverse effects on the central nervous system, manifesting as headache, dizziness, numbness, tremors, and vision problems. Chronic cyanide exposure may also affect the thyroid gland, irritate the eyes and skin, and affect the cardiovascular and respiratory systems. The US EPA (2009b) lists cyanide as “not classifiable” as to human carcinogenicity (group D).

Diesel exhaust

Usage, emissions sources and atmospheric processes

Nonroad and onroad diesel engines emit a complex gas-particle mixture of both known and unknown compounds that include confirmed carcinogens like benzene (US EPA, 2002). The physicochemical transformation and removal of diesel exhaust species in the atmosphere varies across the spectrum of components. As with the other toxic species listed here, reactions with the hydroxyl radical are an important sink for many components of diesel exhaust (US EPA, 2002). Indeed some of the components of diesel exhaust have been listed here already. Primary diesel particles, which are made up of soot and organic carbon, provide a core upon which other inorganic and organic species can adsorb. These particles are subject to gas-particle mass transfer, aqueous phase chemistry, and deposition processes, and due to their size, may be transported significant distances away from the source area (US EPA, 2002). In addition, diesel PM can contribute to visibility reduction and climate change (CARB, 2005).

Health impacts

Diesel exhaust has been linked to increased cancer risk in addition to non-cancer risks, including adverse effects on the respiratory and cardiovascular systems that can especially affect vulnerable segments of the population (US EPA, 2002). Short-term exposures may cause lung irritation and exacerbation of asthma or allergies, while chronic exposures may result in lung cancer or lung damage (US EPA, 2002). Though the US EPA has not classified diesel engine exhaust as a carcinogen, the US EPA does consider it “likely to be carcinogenic to humans by inhalation from environmental exposures” (US EPA, 2012). NJDEP ranks diesel exhaust exposure as having the highest cancer risk among air toxics (NJ DEP 2011). The US EPA does not apply a cancer risk factor for diesel exhaust in NATA.

Ethylbenzene

Usage, emissions sources and atmospheric processes

Ethylbenzene is used in the production of styrene, fuels, in asphalt and naphtha, and as a solvent. Exposure to ethylbenzene may occur through occupational contact or through the use of consumer products, gasoline, pesticides, solvents, carpet glues, varnishes, paints, and tobacco smoke (US EPA, 2009b).

Health impacts

Short-term exposure can result in eye and throat irritation or dizziness. In human studies of chronic exposure by inhalation, there have been conflicting results on the effects on blood; while animal studies have seen effects on the blood, liver, and kidneys. The US EPA lists ethylbenzene as “not classifiable” for human carcinogenicity (group D) (US EPA, 2009b).

Formaldehyde

Usage, emissions sources and atmospheric processes

Mobile sources, incinerators, industrial facilities, and power plants are major emitters of formaldehyde (US EPA, 2009b), and it is also present in tobacco smoke. Formaldehyde is used in the manufacturing of particleboard and in production of other chemicals. Atmospheric formaldehyde concentrations are influenced by direct emissions as well as chemical production and removal. Formaldehyde concentrations tend to be higher in the summer months due to higher temperatures, photolysis rates, and biogenic emissions (Luecken et al., 2006). In an annual simulation of air toxic species, Luecken et al. (2006) found that photochemical production accounted for more than half of the modeled total aldehyde concentrations. While direct emissions of aldehydes are important, especially in urban areas during the winter, photochemical production may have a greater impact on ambient concentrations (Luecken et al., 2006).

Health impacts

Short-term exposure by inhalation may lead to eye, nose, and throat irritation. Short-term exposure to a high level of formaldehyde may result in coughing, wheezing, chest pain, and bronchitis. Chronic exposure may also lead to respiratory and/or skin irritation as well as lung or nasopharyngeal cancers. The US EPA (2009b) has classified formaldehyde as a probable human carcinogen (group B1).

Lead compounds

Usage, emissions sources and atmospheric processes

Lead was once used as an additive in gasoline, but since the phase-out of leaded gasoline, atmospheric levels have significantly decreased. Lead is currently used in the production of batteries, metal products, paints, and glazes. Lead can also be released into the atmosphere from the combustion of coal, oil, and solid waste and during iron and steel manufacturing. Lead is contained in tobacco smoke, in pipes, and in lead-based paints

(US EPA, 2009b). It can be found in an array of different compounds, each with different physical properties. Lead has an ambient residence time of 7 to 30 days in the atmosphere and is removed by deposition (Seinfeld and Pandis, 1998).

Health impacts

Exposure can occur through breathing contaminated air or ingesting lead-containing dust or paint chips. Children are at particularly high risk of being exposed to lead through ingestion. Short term exposure of high levels of lead may lead to brain and kidney damage and death. At lower levels, short-term lead exposure can also have adverse effects on the gastrointestinal system. Chronic lead exposure can lead to anemia and affect the central nervous system, blood pressure, kidney function, and metabolism of vitamin D. Children may be affected by diminished cognitive ability and stunted growth if suffering from chronic lead exposure. In addition, lead exposure may lead to adverse reproductive effects in men and women. The US EPA (2009b) has classified lead as a probable human carcinogen (group B2).

Manganese compounds

Usage, emissions sources and atmospheric processes

Manganese is found naturally throughout the environment. It is used in steel manufacturing, and manganese compounds are also used in chemical production, batteries, matches, fertilizer, animal feed, vitamins, glazes, and fireworks. Manganese can be emitted into the atmosphere from iron and steel manufacturing plants, coke ovens, and power plants (US EPA, 2009b). Manganese compounds are solids, and atmospheric manganese is generally found in small particles. In clouds and fogs, manganese may serve as a catalyst in the aqueous sulfur (IV) oxidation to sulfur (VI), leading to the production of particulate sulfate (Seinfeld and Pandis, 1998).

Health impacts

While manganese is essential for healthy body function at low levels, chronic exposure to high levels by inhalation may adversely affect the central nervous system. Chronic exposure may result in slower reaction time, impaired coordination, weakness, tremors, and speech and psychological disturbances. The respiratory system may also be adversely affected. There are no reports of effects associated with short-term acute exposure. The US EPA (2009b) lists manganese as “not classifiable” for human carcinogenicity (group D).

Methyl bromide

Usage, emissions sources and atmospheric processes

Natural sources account for a significant percentage of methyl bromide emissions, with oceans as the largest natural source (Seinfeld and Pandis, 1998). Methyl bromide is used primarily as a fumigant, methylating agent, and solvent (US EPA, 2009b; ATSDR, 2007). Methyl bromide is found throughout the atmosphere as a result of both biogenic and anthropogenic sources. It has an atmospheric lifetime of a little over a year due to

destruction by the hydroxyl radical, hydrolysis, and deposition (Seinfeld and Pandis, 1998).

Health impacts

Methyl bromide is very toxic. Most exposure is via inhalation or absorption through the skin, and methyl bromide, which is a gas at room temperature, can penetrate most protective clothing and skin (US EPA, 2009b; ATSDR, 2007). Short-term, acute exposures may severely injure the lungs, and inhaling high methyl bromide concentrations may lead to pulmonary edema. Additionally, acute exposures may result in headaches, dizziness, fainting, weakness, confusion, speech and vision effects, numbness, twitching, and tremors. Very high exposures may lead to paralysis or convulsions. It is an irritant to the skin, eyes, and mucous membranes. Chronic exposure may cause mild neurological impairment. The US EPA (2009b) lists methyl bromide as “not classifiable” for human carcinogenicity (group D).

Methyl chloride

Usage, emissions sources and atmospheric processes

Methyl chloride is emitted by natural and anthropogenic sources. It is used in chemical plants and is also emitted from the ocean and biomass burning. It can be found in tobacco smoke (US EPA, 2009b). Methyl chloride is the dominant halogen compound found in the atmosphere and is a continuous natural source of chlorine to the stratosphere. It has an atmospheric lifetime on the order of two years, and the ocean is its largest emissions source on a global level. The primary atmospheric sink of methyl chloride is its reaction with the hydroxyl radical; though it may also be broken down by photolysis. With the release of an active chlorine atom, these reactions can lead to stratospheric ozone depletion (Seinfeld and Pandis, 1998).

Health impacts

Short-term exposures to high levels of methyl chloride may result in severe neurological effects, such as convulsions or coma. Dizziness, double vision, slurred speech, nausea, vomiting, and heart rate, liver, and kidney effects may also occur. While the effects of chronic exposure of methyl chloride on humans are not clear, animal studies point to possible adverse effects on the liver, kidneys, spleen, and central nervous system. The US EPA (2009b) lists methyl chloride as “not classifiable” for human carcinogenicity (group D).

Naphthalene

Usage, emissions sources and atmospheric processes

Naphthalene is emitted during the combustion of coal and oil, can be found in tobacco smoke, and is used in mothballs. Its primary use is in the production of phthalic anhydride, which is an important industrial chemical used mainly in the plastics industry (US EPA, 2009b; OEHA, 2000). The most significant sources of naphthalene in an urban environment are mobile sources and residential heating (Martinez et al., 2004).

Naphthalene is the simplest and most volatile polycyclic aromatic hydrocarbon (PAH) and is the most abundant PAH in urban areas. The lifetime of naphthalene is shorter than one day mainly due to the reaction of naphthalene with the hydroxyl radical (Martinez et al., 2004).

Health impacts

Short-term exposure can lead to hemolytic anemia, liver damage, and neurological damage. Cataracts have been seen in workers with acute exposure to naphthalene. Additional symptoms of acute exposure can include headache, nausea/vomiting, confusion, convulsions, or coma. Long-term impacts include cataracts and retina damage, and infants may be born with hemolytic anemia if their mothers inhaled or ingested naphthalene while pregnant. The US EPA (2009b) has classified naphthalene as a possible human carcinogen (group C).

Toluene

Usage, emissions sources and atmospheric processes

Toluene is added to gasoline to improve octane ratings. It is also used as a solvent and in the production of benzene, polymers, and other chemicals. Toluene may be released into the air from motor vehicle exhaust and through the use of household products. Toluene is active in ozone chemistry and can contribute to the formation of secondary organic aerosol (Seinfeld and Pandis, 1998).

Health impacts

Adverse effects on the central nervous system may result from both short-term and chronic exposures. Fatigue, headache, and nausea may result from lower exposures; exposures to higher levels of toluene can lead to central nervous system depression and death. Cardiac arrhythmia may also result from acute exposure. Chronic exposure may lead to central nervous system depression, irritation of the eyes and upper respiratory tract, and mild kidney and liver effects. Toluene exposure in pregnant women may also lead to adverse developmental effects. The US EPA lists toluene as “not classifiable” for human carcinogenicity (group D) (US EPA, 2009b).

Xylenes

Usage, emissions sources and atmospheric processes

Mixed xylenes (m-xylene, p-xylene, and o-xylene isomers) are emitted from industrial and mobile sources. Xylenes are used to make ethylbenzene, are found in gasoline, and used as solvents. Xylenes are active in ozone chemistry and can contribute to the formation of secondary organic aerosol. Xylene isomers' reactions with hydroxyl radical are a major atmospheric sink; m-xylene has a lifetime on the order of seven hours (Seinfeld and Pandis, 1998).

Health impacts

Short-term exposure can lead to irritation of the eyes, nose, throat, skin, or gastrointestinal system. It may also result in neurological effects like slowed reaction time and impaired short-term memory and balance. Chronic exposure leads to adverse neurological effects like headache, dizziness, fatigue, anxiety, tremors, impaired short-term memory and concentration, and may also lead to adverse effects on the lungs, heart, and kidneys. The US EPA (2009b) lists mixed xylenes as “not classifiable” for human carcinogenicity (group D).

2.4. Grouping air toxic species

The air toxics species examined in this analysis can be divided into three main groups linked according to common spatial and temporal behavior:

Group A: Species that are directly emitted, readily removed by direct and/or indirect photolysis or other chemical processes in the atmosphere but not significantly affected by atmospheric production (e.g., benzene, 1,3-butadiene, xylenes, toluene, ethylbenzene)

Group B: Species that have limited emissions and are very slowly removed in the atmosphere, are well-mixed, and have long atmospheric lifetimes (e.g., carbon tetrachloride)

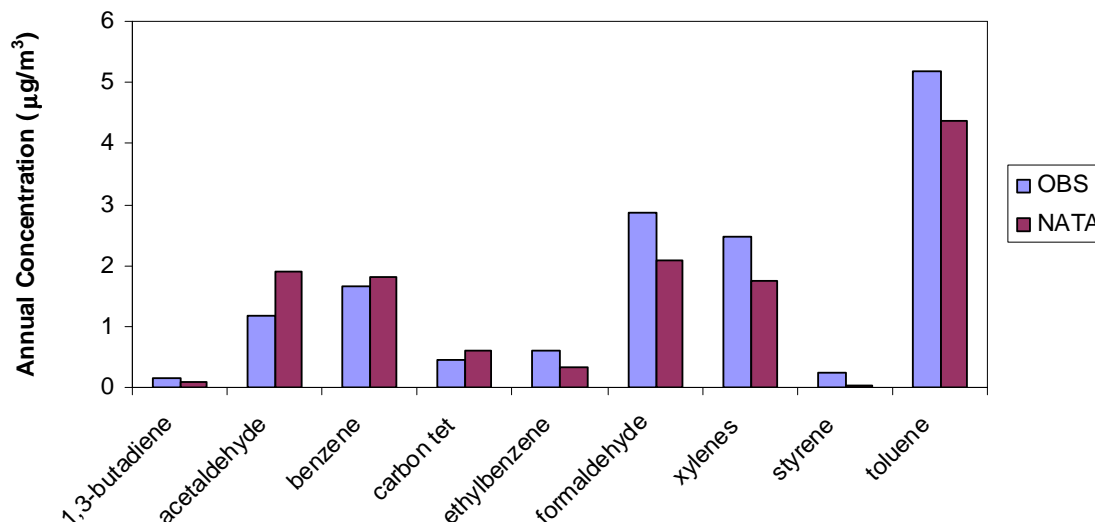
Group C: Species that are directly emitted, are readily removed by direct and/or indirect photolysis in the atmosphere, and are also produced by other species (e.g., formaldehyde, acetaldehyde, acrolein)

These groupings will be used throughout this report.

2.5. Comparison of NATA data with monitoring data

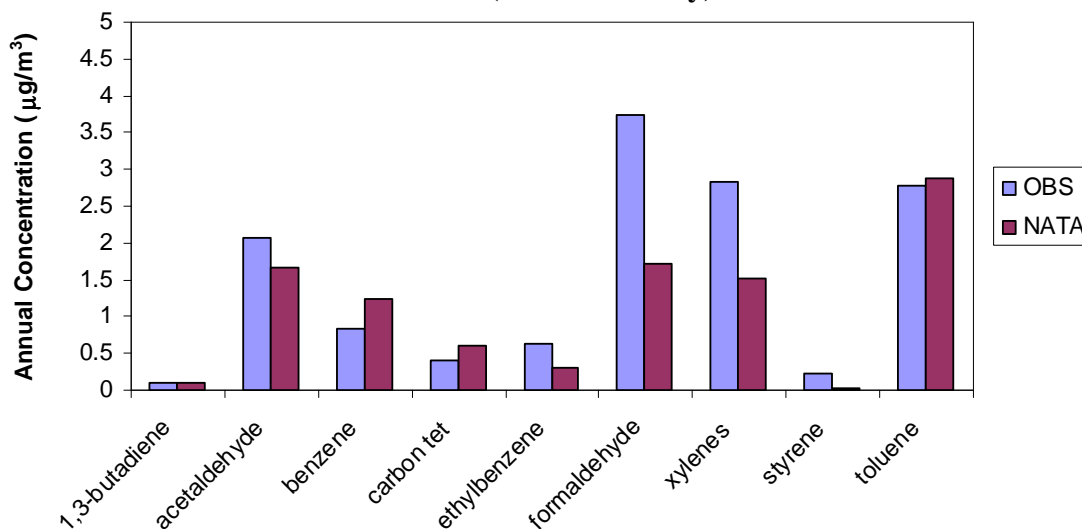
For consistency with CMAQ modeling, which was only performed for 2002, only NATA 2002 data are compared against CMAQ results. For select species, measured concentrations at Camden, Chester, Elizabeth, and New Brunswick (see Section 3.1) were compared with county-averaged NATA ambient concentrations for 2002. It is important to note that while the observations represent concentrations at a single point, the NATA 2002 ambient concentrations represent the annual average concentrations of an entire county. In spite of this, the comparison of the two datasets at Camden and Elizabeth match well for most species, and the relative concentration levels between species are similar for NATA county-averaged ambient concentrations and measurements at the Chester and New Brunswick sites. The largest concentration differences are not much more than a factor of 2 from one another. This magnitude of difference is reasonable given that the comparison is between a county averaged concentration and a concentration measured at a single point. Comparisons are presented in Figure 2-4 through Figure 2-7.

Figure 2-4. Comparison of NATA 2002 predicted and observed concentrations at Camden (Camden County)



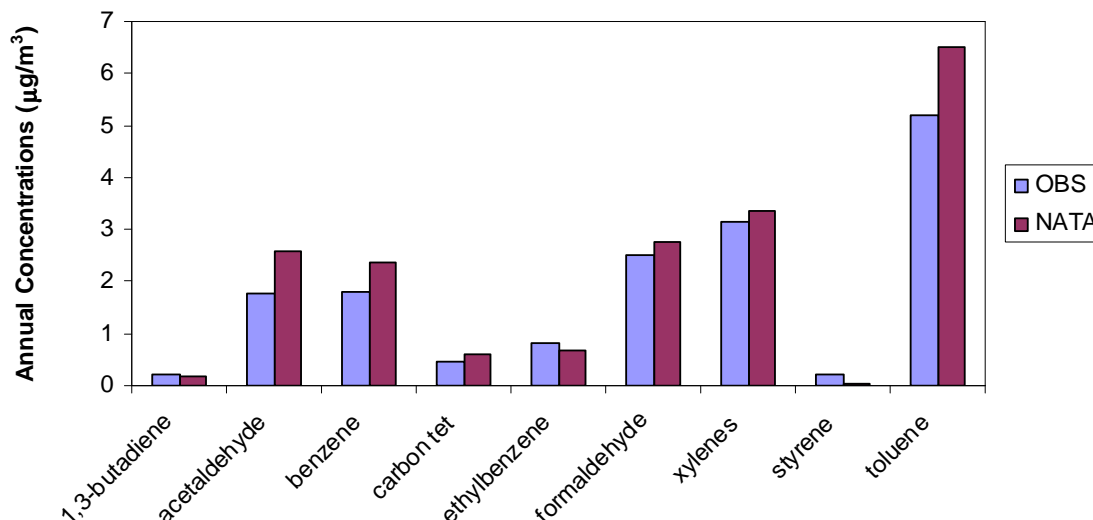
Note: Monitoring data are annual average concentrations from the NJ Air Toxics site at Camden. NATA 2002 data are for Camden County.

Figure 2-5. Comparison of NATA 2002 predicted and observed concentrations at Chester (Morris County)



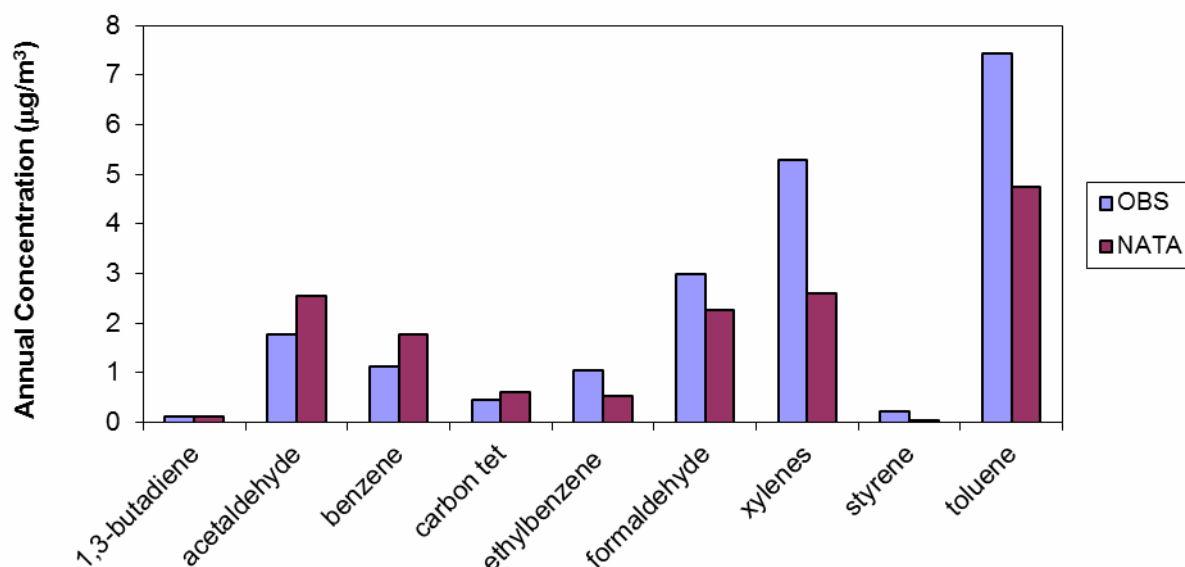
Note: Monitoring data are annual average concentrations from the NJ Air Toxics site at Chester. NATA 2002 data are for Morris County.

Figure 2-6. Comparison of NATA 2002 predicted and observed concentrations at Elizabeth (Union County)



Note: Monitoring data are annual average concentrations from the NJ Air Toxics site at Elizabeth. NATA 2002 data are for Union County.

Figure 2-7. Comparison of NATA 2002 predicted and observed concentrations at New Brunswick (Middlesex County)



Note: Monitoring data are annual average concentrations from the NJ Air Toxics site at New Brunswick. NATA 2002 data are for Middlesex County.

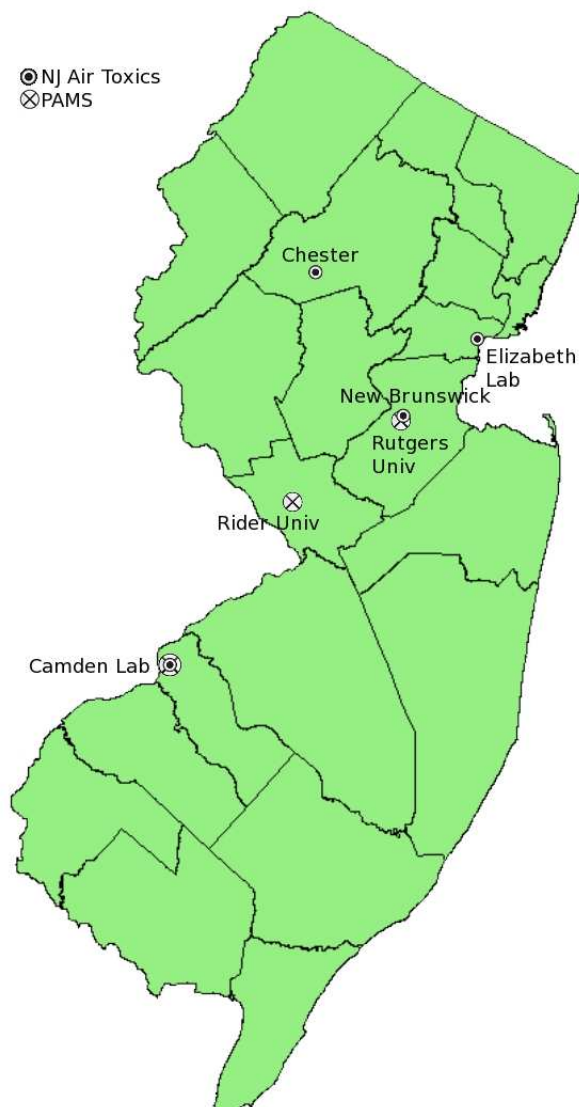
Based on these comparisons between the observed and predicted annual average concentrations of key air toxics, we conclude that the NATA 2002 model performed reasonably well.

3. ANALYSIS OF AIR TOXICS MONITORING DATA

Analysis of ambient monitoring data for individual air toxics species in New Jersey and surrounding states provides a real-world understanding of air toxics levels that exist in and around New Jersey. The data also provide a basis for identifying geographic and temporal patterns of air toxics levels. Prior to this report, analysis of the air toxics data collected by New Jersey's air toxics network had generally been limited to basic summary statistics on annual averages and inter-site comparisons. Additional analyses on temporal and spatial variability, characterization of localized impacts, and analysis of nearby sources will give a better understanding of the nature of air toxics in the region and allow better comparison to modeling data, thus aiding the development of exposure mitigation strategies.

The analysis presented here is focused on those mobile source air toxics that are

Figure 3-1. Air toxics monitoring sites in New Jersey



most prevalent in the state and pose the largest public health risk (acetaldehyde, benzene, formaldehyde, and 1,3-butadiene), though data for all other monitored air toxics compounds were also reviewed. This section presents a summary of findings from this analysis.

3.1. Air toxics monitoring networks

Two monitoring networks were used to gather air toxics monitoring data. NJDEP has measured a group of toxic volatile organic compounds (VOCs) in Camden as part of the Urban Air Toxics Monitoring Program (UATMP) since 1989. Three other sites were established to monitor air toxics in the state: Elizabeth in 1999, and Chester and New

Table 3-1. Relevant parameters measured for the PAMS program

Param.	CAS No.	Chemical Species	Param.	CAS No.	Chemical Species
43202	74-84-0	Ethane	43291	565-59-3	2,3-Dimethylpentane
43203	74-85-1	Ethylene	43249	589-34-4	3-Methylhexane
43204	74-98-6	Propane	43250	540-84-1	2,2,4-Trimethylpentane
43205	115-07-1	Propylene	43232	142-82-5	<i>n</i> -Heptane
43214	75-28-5	Isobutane	43261	108-87-2	Methylcyclohexane
43212	106-97-8	<i>n</i> -Butane	43252	565-75-3	2,3,4-Trimethylpentane
43206	2122-48-7	Acetylene	45202	108-88-3	Toluene
43216	93196-02-2	<i>trans</i> -2-Butene	43960	592-27-8	2-Methylheptane
43213	9003-28-5	1-Butene	43253	589-81-1	3-Methylheptane
43217	590-18-1	<i>cis</i> -2-Butene	43233	111-65-9	<i>n</i> -Octane
43282	287-92-3	Cyclopentane	45203	100-41-4	Ethylbenzene
43221	92046-46-3	Isopentane	45109	179601-23-1	<i>m,p</i> -Xylenes
43220	8031-35-4	<i>n</i> -Pentane	45220	9003-53-6	Styrene
43226	68956-55-8	<i>trans</i> -2-Pentene	45204	95-47-6	<i>o</i> -Xylene
43224	8029-09-2	1-Pentene	43235	111-84-2	<i>n</i> -Nonane
43227	627-20-3	<i>cis</i> -2-Pentene	45210	98-82-8	Isopropylbenzene
43244	75-83-2	2,2-Dimethylbutane	45209	103-65-1	<i>n</i> -Propylbenzene
43284	79-29-8	2,3-Dimethylbutane	45212	620-14-4	<i>m</i> -Ethyltoluene
43229	107-83-5	2-Methylpentane	45213	622-96-8	<i>p</i> -Ethyltoluene
43230	96-14-0	3-Methylpentane	45207	108-67-8	1,3,5-Trimethylbenzene
43243	78-79-5	Isoprene	45211	611-14-3	<i>o</i> -Ethyltoluene
98040	763-29-1	2-Methyl-1-pentene	45208	95-63-6	1,2,4-Trimethylbenzene
42231	110-54-3	<i>n</i> -Hexane	43238	124-18-5	<i>n</i> -Decane
43262	96-37-7	Methylcyclopentane	45225	526-73-8	1,2,3-Trimethylbenzene
43247	108-08-7	2,4-Dimethylpentane	45218	141-93-5	<i>m</i> -Diethylbenzene
45201	71-43-2	Benzene	45219	105-05-5	<i>p</i> -Diethylbenzene
43248	110-82-7	Cyclohexane	43954	1120-21-4	<i>n</i> -Undecane
43263	591-76-4	2-Methylhexane	43102	-	Total non-methane organic compounds (NMOC)

Brunswick in 2001. These sites are collectively referred to as the New Jersey Air Toxics network throughout this report. In addition to the New Jersey Air Toxics network, there are three Photochemical Assessment Monitoring Stations (PAMS) at Rider University, Rutgers University, and Camden. A map of these monitors is provided in Figure 3-1. Additional details for the New Jersey Air Toxics Network and the PAMS network are provided in Sections 3.1.1 and 3.1.2, respectively.

3.1.1. New Jersey Air Toxics network

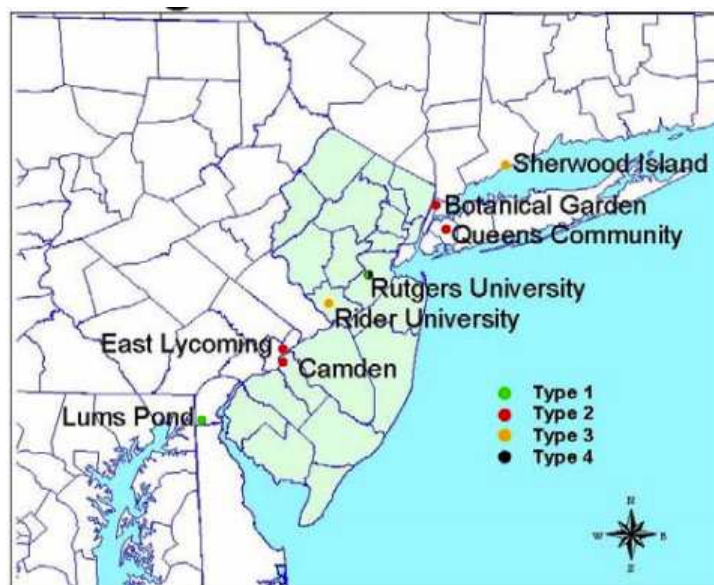
The four air toxics monitoring stations in New Jersey collect samples every six days, according to the TO-15 analysis method for VOCs, and the TO-11A method for formaldehyde and other carbonyl compounds. The TO-15 analysis method involves use of a steel canister for collecting air samples for VOC analysis, and the TO-11A method uses a DNPH cartridge for measurement of carbonyls.

3.1.2. Photochemical Assessment Monitoring Stations (PAMS)

PAMS were established based on February 1993 revisions to Title 40 Part 58 of the Code of Federal Regulations (40 CFR Part 58) to expand the existing networks in ozone nonattainment areas. PAMS were specifically designed to monitor volatile organic compounds (VOCs), which are precursor compounds to ozone formation. Because many VOCs are classified as air toxics, the PAMS network is a valuable tool for assessing the levels of some air toxics at an hourly average timescale. There are three PAMS network sites in New Jersey. The Rider University PAMS was established as a Type 3 site, meaning it is likely to experience maximum ozone concentrations as it is downwind from major precursor sources. The Camden Lab PAMS is categorized as a Type 2 site, indicating that it is likely to experience maximum ozone precursor concentrations due to nearby sources. The Rutgers University PAMS is a Type 1 (upwind) site from the New York City area. The PAMS database provides hourly data on several air toxics that can be used to analyze temporal (including diurnal profiles) and spatial variability of certain gas-phase air toxics in the state. These sites are only operational during the summer ozone season. A list of VOC compounds measured by the PAMS network is presented in Table 3-1, with each compound's applicable US EPA Air Quality System (AQS) parameter code and Chemical Abstract Service (CAS) number.

3.1.3. Measurements from neighboring states

Air toxics measurements are also collected at sites in neighboring states. The air toxics data from these regional sites were downloaded from the AQS Data Mart and analyzed using Excel to generate plots demonstrating the annual, quarterly, and diurnal trends of species at these locations. The sites included: Sherwood Island, CT; Botanical Gardens, NY; Community College, NY; East Lycoming, PA; and Lums Pond, DE. A map showing the locations of air toxics monitoring sites in relation to those operating in New Jersey is presented in Figure 3-2 (reproduced from NJDEP, 2003). NESCAUM focused on four air toxics from Group A (benzene, xylene, toluene, and ethylbenzene) and two air toxics from Group C (formaldehyde and acetaldehyde) for this comparison. Note that not all species were measured during all years of interest at these sites. Also note that data from these sites were not used during the model evaluation. Results of this regional comparison are discussed in context in Section 3.4.4.

Figure 3-2. Air toxics monitoring sites in states neighboring New Jersey

Source: NJDEP, 2003. Reproduced from the original.

3.2. Monitoring data sources and processing

The data for select air toxics species were retrieved for all New Jersey sites for years 2000-2009 from the US EPA's AQS Data Mart³ and also from NJDEP staff. The downloaded XML files were organized by site, species, and measurement type and saved into separate comma separated value (CSV) files.

Table 3-2 contains a description of data files received from NJDEP. For the data analyses presented in Section 3.4 and Appendix A, NESCAUM used the data from the AQS Data Mart (Table 3-3) rather than NJDEP files as the former included information on the minimum detection limit (MDL) for each measurement and had a uniform format. Minimum detection limits are defined as "the lowest concentrations at which laboratory equipment have been experimentally determined to reliably quantify concentrations of selected pollutants to a specific confidence level" (Eastern Research Group, 2008). Such data should be used with caution due to the increasing uncertainty at or below the MDL. NESCAUM used data from NJDEP to spot check the files downloaded from the AQS Data Mart. NESCAUM developed Perl scripts to read the CSV data files and calculate averages, replaced concentrations below the MDL with half the MDL, counted the number of replacements of below MDL values, and generated data files to import into Excel.

³ US EPA, Air Quality System (AQS) Data Mart, <http://www.epa.gov/ttn/airs/aqsdatamart/>

Table 3-2. Data files obtained from NJDEP

File Name	Site	Year	Compound(s)
toxics 1.mdb	All sites	2004-2008	VOCs and carbonyls
data sumary.xls	All sites	1995-2008	Summary of available data
Camdencarb96.xls	Camden Lab	1996	Carbonyls
Camdencarb97.xls	Camden Lab	1997	Carbonyls
Camdencarb98.xls	Camden Lab	1998	Carbonyls
Camdencarb99.xls	Camden Lab	1999	Carbonyls
Camdencarb00.xls	Camden Lab	2000	Carbonyls
Camdencarb01.xls	Camden Lab	2001	Carbonyls
cam2002alde.xls	Camden Lab	2002	Carbonyls
camden carbs 2003.xls	Camden Lab	2003	Carbonyls
camden1994-2000-vocs.xls	Camden Lab	1994-2000	VOCs
cam-vocs99.xls	Camden Lab	1999	VOCs
Cam2001Rawvoc.xls	Camden Lab	2001	VOCs
cam2002rawvoc.xls	Camden Lab	2002	VOCs
camden voc 2003.xls	Camden Lab	2003	VOCs
Chest2001alde2.xls	Chester	2001	Carbonyls
che2002alde.xls	Chester	2002	Carbonyls
chester carbs 2003.xls	Chester	2003	Carbonyls
chester2001rawvoc.xls	Chester	2001	VOCs
che2002rawvoc.xls	Chester	2002	VOCs
chestervocs 2003.xls	Chester	2003	VOCs
eliz-carbs-sum-2000.xls	Elizabeth	2000	Carbonyls
eliz2001alde2.xls	Elizabeth	2001	Carbonyls
eliz2002alde.xls	Elizabeth	2002	Carbonyls
elizabeth carbs 2003.xls	Elizabeth	2003	Carbonyls
eliz-voc-sum-2000.xls	Elizabeth	2000	VOCs
eliz2001rawvoc.xls	Elizabeth	2001	VOCs
eliz2002vocraw.xls	Elizabeth	2002	VOCs
elizabeth voc 2003.xls	Elizabeth	2003	VOCs
NewBruns2001alde2.xls	New Brunswick	2001	Carbonyls
NewB2002alde.xls	New Brunswick	2002	Carbonyls
NewBrunswick carbs 2003.xls	New Brunswick	2003	Carbonyls
NewBruns2001rawvoc.xls	New Brunswick	2001	VOCs
NewB2002rawvoc.xls	New Brunswick	2002	VOCs
NewBrunswick voc2003.xls	New Brunswick	2003	VOCs
warren county 2003.xls	Warren County ¹	2003	VOCs

Source: Joshua Ray, NJDEP

1. The Warren County Air Monitoring Program (WCAMP) monitored several parameters, including selected volatile organic compounds, for several years. Additional information about this monitor is available at <http://dsmcap.com/seps/about.htm>.

Table 3-3. Data obtained from the AQS Data Mart

Species	Sites	Years	Time Resolution
Acetaldehyde	Camden	2000-2008	24-hour, limited 3-hour
	Chester	2001-2009	24-hour
	Elizabeth	2000-2009	24-hour
	New Brunswick	2001-2009	24-hour
Acrolein	Camden	2005-2008	24-hour
	Chester	2005-2009	24-hour
	Elizabeth	2005-2009	24-hour
	New Brunswick	2005-2009	24-hour
Benzene	Camden	2000-2008	24-hour, limited 1-hour
	Chester	2001-2009	24-hour
	Elizabeth	2000-2009	24-hour
	New Brunswick	2001-2009	24-hour
1,3-Butadiene	Camden	2000-2008	24-hour
	Chester	2001-2009	24-hour
	Elizabeth	2000-2009	24-hour
	New Brunswick	2001-2009	24-hour
Carbon Tetrachloride	Camden	2000-2008	24-hour
	Chester	2001-2009	24-hour
	Elizabeth	2000-2009	24-hour
	New Brunswick	2001-2009	24-hour
Ethylbenzene	Camden	2000-2008	24-hour, limited 1-hour
	Chester	2001-2009	24-hour
	Elizabeth	2000-2009	24-hour
	New Brunswick	2001-2009	24-hour
Formaldehyde	Camden	2000-2008	24-hour, limited 3-hour
	Chester	2001-2009	24-hour
	Elizabeth	2000-2009	24-hour
	New Brunswick	2001-2009	24-hour
<i>m,p</i> -Xylene	Camden	2000-2008	24-hour, limited 1-hour
	Chester	2001-2009	24-hour
	Elizabeth	2000-2009	24-hour
	New Brunswick	2001-2009	24-hour
<i>o</i> -Xylene	Camden	2000-2008	24-hour, limited 1-hour
	Chester	2001-2009	24-hour
	Elizabeth	2000-2009	24-hour
	New Brunswick	2001-2009	24-hour
Toluene	Camden	2000-2008	24-hour, limited 1-hour
	Chester	2001-2009	24-hour
	Elizabeth	2000-2009	24-hour
	New Brunswick	2001-2009	24-hour

Source: US EPA, AQS Data Mart.

NESCAUM calculated annual, quarterly, seasonal, and diurnal averages.⁴ No data-completeness requirements were imposed on the data comprising the annual, quarterly, or seasonal averages for any species or site.

Most of the figures in Section 3.4.4 and in Appendices A and B were generated using these data files in Excel. The programs also matched measurement days to facilitate comparison between the PAMS and NJ Air Toxics networks described in Appendix A. The PAMS to NJ Air Toxics network scatter plots in Appendix A were generated using R statistical software.

Annual, quarterly, and seasonal (summer and winter) concentrations were calculated for benzene, acetaldehyde, acrolein, 1,3-butadiene, formaldehyde, carbon tetrachloride, xylenes, toluene, and ethylbenzene. No data completeness requirements were imposed on the data comprising the annual, quarterly, or seasonal averages for any species or site. PAMS data were extracted for benzene, toluene, xylenes, ethylbenzene, formaldehyde, and acetaldehyde. The PAMS data were used to estimate diurnal behavior (weekday/weekend). Twenty-four hours of data were required for a day's measurements to be included in the averages representing "typical" diurnal behavior in Section 3.4.2. Excel was used to create the figures in this section. The scripts also matched measurement days to facilitate comparisons between the PAMS and NJ Air Toxics networks.

NESCAUM conducted the following analyses using the monitoring data: analysis of spatial characteristics including inter-site comparisons and analysis of local emission sources or transport patterns; analysis of temporal characteristics including descriptions of annual averages and seasonal variation; and examination of diurnal profiles when PAMS data were available.

3.3. Sampling methods and uncertainty

As with all ambient monitoring, there can be uncertainty in the observed measurements due to environmental variability and monitoring error in sample collection and laboratory analysis. For the majority of data in the UATMP network, it was determined that environmental variability, especially temporal, accounts for the overall variability observed; however, monitoring error becomes more significant at lower ambient concentrations (Bortnick and Stetzer, 2002). Given that NJDEP has maintained and operated the air toxics and PAMS sites in New Jersey for many years, there are robust data quality assurance and control procedures in place. Despite carefully following procedures, it is still possible to have monitoring uncertainty as evidenced by acrolein growth in monitoring canisters due to insufficient canister cleaning practices (Jones, 2010).

The New Jersey PAMS sites use gas-chromatograph (GC) with flame ionization detector (FID) technology with thermal desorption. Sampling occurs over the first 40 minutes of each hour followed by measurement. Additional details of the technologies,

⁴ Quarter 1 (Q1) includes January, February, and March; Q2, April, May, and June; Q3, July, August, and September; and Q4, October, November, and December. Seasonal averages are for summer (June, July, and August) and winter (January, February, and December of the previous year).

procedures, maintenance, and operation of systems used for sampling and measuring air toxics concentrations are presented in the Appendix A of the Quality Assurance Project Plan (QAPP).

3.4. Monitoring data results

This section presents a summary of data analysis of New Jersey Air Toxics and PAMS monitoring data. The species examined in this analysis are divided into the three main groups described in Section 2.4.

Data for certain compounds collected using the standard method have been subject to additional data quality scrutiny in recent years (US EPA, 2010a). Monitoring data for acrolein in particular have been determined by the US EPA to be an issue across its monitoring program (US EPA, 2010b). Communication from Charles Pietarinen of NJDEP indicated that the quality of the data for formaldehyde and acetaldehyde monitored through the NJ Air Toxics program are not well understood, that the quality of acrolein data was poor, and that the quality of 1,3-butadiene data varied (Pietarinen, 2011). The analysis presented here should be understood in light of these data quality problems.

General discussion of multi-year and seasonal trends are examined in Section 3.4.1 using data from the NJ Air Toxics network, and diurnal behavior using the PAMS network data is examined in Section 3.4.2. A comparison between NJ Air Toxics data and PAMS data is presented in Section 3.4.3. Detailed results arranged into the groups are presented in Section 3.4.4.

3.4.1. Summary of New Jersey Air Toxics monitoring data results

This section presents a summary of data analysis of New Jersey Air Toxics monitoring data. The different groups of compounds exhibited different trends during the years considered (2000-2009).

For most species in Group A, there was an overall downward trend in annual average concentrations at most sites. However, for some species at some sites, the data did not clearly point toward a trend. These anomalies may be due to high individual readings from event plumes (a significant toxics release from an industrial facility, for instance), or could be due to an across-the-board increase in emissions of those compounds. The Elizabeth site typically had the highest concentrations of these species, followed by Camden, New Brunswick, and Chester.

Seasonal patterns amongst Group A compounds were not uniform. For benzene and 1,3-butadiene, the summer concentrations often exceeded winter concentrations of the same year. For xylenes, toluene, and ethylbenzene, the relationship between seasonal concentrations was more variable.

Carbon tetrachloride is the only species examined in Group B. At all sites, it showed a rough upwards trend from year to year. This upward trend is not surprising given the long lifetime of carbon tetrachloride (decades), even despite the lack of strong domestic emission sources. Because of the relative atmospheric stability of carbon tetrachloride, even small, distant emission sources may be able to sustain and increase concentrations in New Jersey. According to the US EPA (2008), there are no significant

sources of carbon tetrachloride within New Jersey. Nationally, 7 sources accounted for the majority of domestic carbon tetrachloride emissions, and only 34 sources emitted more than 1 ton per year, accounting for 89 percent of domestic emissions. Because there are few emission sources of carbon tetrachloride in and around New Jersey, monitors in the region are influenced by regional, national, and international sources, and therefore all sites had similar concentration levels. Summer concentrations often surpassed winter concentrations for a given year.

Group C compounds' concentrations rose steadily in some cases or were more volatile from year to year. Concentrations between sites were also variable, and sites with the highest concentrations often changed from year to year. Summer concentrations for all Group C compounds often surpassed winter concentrations. Because these compounds are formed in the atmosphere through chemical interaction requiring sunlight and relatively higher temperatures, it is expected that summer concentrations would be higher than those seen in the winter. Because NJDEP indicated that there are data quality issues related to the monitoring data for these species (Pietarinen, 2011), NESCAUM does not have a high degree of confidence to make statements about year-to-year trends or spatial relationships between the sites for these species.

3.4.2. Summary of New Jersey PAMS data results

There are limited data capturing diurnal behavior, but for those species with data, there are similarities in diurnal behavior amongst species within a group. Group A compounds (benzene, xylenes, toluene, and ethylbenzene) show early morning peaks, minimums in the early afternoon, and rising concentrations starting in the early evening. The morning peak may be due to early morning emissions, and the afternoon low is likely due to indirect photolysis and a rising atmospheric mixing height. The rise in concentration following the afternoon trough may be due to a fresh influx of emissions that are much more slowly depleted at night along with a lowering mixing height.

Carbon tetrachloride is not among the species measured at the PAMS locations (see Table 3-1), and therefore Group B is not represented in the PAMS data.

Concentrations of Group C compounds (formaldehyde and acetaldehyde) rise from early morning to an early afternoon peak, a peak likely associated with increased photochemical production. These species' concentrations are gradually reduced until the early morning when fresh emissions are released into the atmosphere and photochemical production leads to higher concentrations. Because NJDEP indicated that there are data quality issues related to the monitoring data for these species (Pietarinen, 2011), NESCAUM does not have a high degree of confidence to make statements about year-to-year trends or spatial relationships between the sites for these species.

3.4.3. Summary of PAMS and NJ Air Toxics data comparisons

While year-round 24-hour samples are typically collected at the New Jersey Air Toxics measurement sites, hourly (or 3-hour average, in the case of carbonyls) samples are collected at PAMS network sites during the summer ozone season. Because some compounds measured by PAMS sites are also measured by the NJ Air Toxics sites, PAMS may be compared with nearby NJ Air Toxics measurements and used to investigate the diurnal patterns for those compounds.

PAMS and NJ Air Toxics data were also examined together to determine if collocated and concurrent PAMS and NJ Air Toxics measurements were comparable. Limited data comparisons were made for formaldehyde, acetaldehyde, benzene, toluene, xylenes, and ethylbenzene. Daily NJ Air Toxics and 24-hour average PAMS measurements at the Camden Lab site were compared on days when there were data for both samplers. Only days with at least 75 percent PAMS data completeness (18 or more hours) were used in the comparison. Time series and scatter plots were generated for benzene, toluene, xylenes, and ethylbenzene, and two different methods of linear regression (“ordinary least squares” and the less outlier-sensitive “least trimmed squares”) were employed to fit the data. While there was significant scatter in the data, the slope of the regression lines (NJ Air Toxics vs. PAMS) estimated for benzene, toluene, and ethylbenzene were between 0.8 and 1.1 for both methods. The slope of the trend line for xylenes was 0.6-0.7. Because xylenes are composed of three isomers (*o*-xylene, *m*-xylene, and *p*-xylene) and *o*-xylene is more likely than benzene or toluene to have concentrations below the reported MDLs, this may have affected the PAMS-NJ Air Toxics comparison for xylenes. While the slopes of the trend lines are not very far from 1, there are many days for which the NJ Air Toxics and PAMS data diverge significantly. This can be due to some of the PAMS averages being calculated with fewer than 24 hours of data or due to differences in monitoring methodologies. A seasonal comparison of NJ Air Toxics and PAMS measurements for the same set of species also indicates that both datasets are comparable for most years. Further details of these comparisons can be found in Appendix A.

3.4.4. Summary of regional air toxics monitoring data

This section presents a summary of data analysis of regional air toxics monitoring data compared to New Jersey Air Toxics and PAMS monitoring data. In general, data from neighboring states exhibit the same temporal trends seen for Group A and C species in New Jersey (Group B was not examined). For example, for benzene (Group A), there is an overall downward annual trend, the highest concentrations are generally in the quarters 1 and 4 (the colder months), and the diurnal pattern shows a morning peak (not as high on the weekend) with an afternoon low. As in New Jersey, there does not appear to be a consistent seasonal trend at regional sites for some species, including acetaldehyde. And, as in New Jersey, formaldehyde monitoring data across the region indicate higher concentrations in the warmer months than in the cooler months, and a diurnal profile featuring high afternoon concentrations. Regional patterns for xylenes, toluene, and ethylbenzene are the same as they are within New Jersey; the annual values show a slight downward trend, there is no clear trend in the quarterly data, and the diurnal plots illustrate a morning maximum and an afternoon minimum, though this effect is less pronounced on weekend days compared to weekdays. By examining the specific air toxics measurements in neighboring states to those located in New Jersey, NESCAUM determined that there is a general consistency in temporal characteristics of these air toxics across the region.

In addition to their temporal characteristics, air toxics patterns and trends are reasonably consistent across the region. Though site-by-site concentration differences underscore the importance of local sources on individual locations, this analysis suggests that many air toxics levels are influenced by regional patterns of air toxics sources. It is

unsurprising that many of the air toxics examined in this report show similar patterns across the region, as they are dominated by mobile sources. Plots of air toxics discussed in this analysis for regional sites are presented in Appendix B.

3.4.5. Detailed monitoring data results

Monitoring results for each species of interest are presented in this section and arranged by the groups described in Section 3.4. Results are presented for annual, quarterly, seasonal, and diurnal averages, as available. For diurnal patterns, because sample sizes are generally small, single events (e.g., a plume with high air toxics concentrations) can have a large effect on the diurnal profile. These occurrences are noted when they occur in the diurnal profiles. Use of median concentrations rather than mean (simple average) concentrations would mitigate this effect.

Group A compounds

Group A compounds are air toxics that are directly emitted, are readily removed by direct and/or indirect photolysis or other chemical processes in the atmosphere, but are not significantly impacted by atmospheric production. Discussions of toluene and ethylbenzene are included here, although they are not on the top lists from NATA for cancer or non-cancer health risks in New Jersey. We include them because they are mobile source air toxics that are measured at both types of monitoring networks and are often measured at levels well above their MDLs.

Benzene

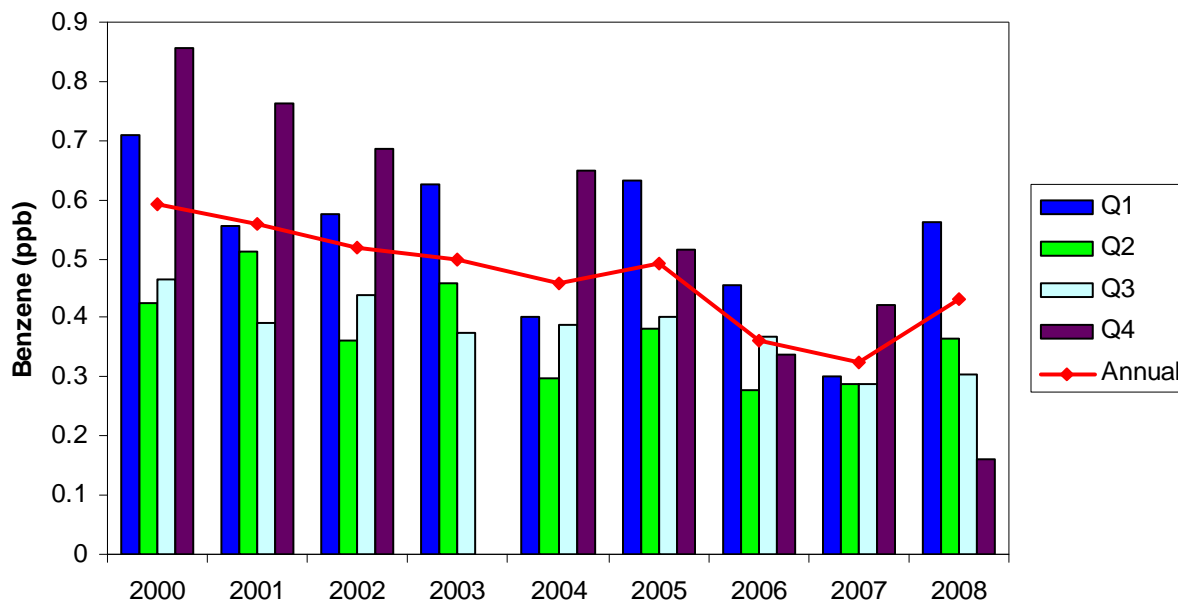
Since 2002, annual benzene concentrations have followed an overall downward trend at New Jersey's long-term air toxics measurement sites with a couple upswings in concentration in 2005 (for three sites) and 2008 (for all sites) (see Figure 3-3 through Figure 3-6). With few exceptions, benzene concentrations were highest in the first and fourth quarters and lowest in the second and third quarters. Benzene is not produced in the atmosphere and is removed through reactions with the hydroxyl radical, which is highest in the warmer months (quarters 2 and 3). The differences in summer and winter concentrations in Figure 3-7 illustrate more clearly the seasonal dependence of ambient benzene levels. As shown in Figure 3-7, winter average concentrations were higher, and often considerably higher, than summer average concentrations for nearly every site and year. The only exception was at Elizabeth in 2008 when the average third quarter concentration was more than double that of all other quarters and the highest quarterly average benzene concentration of all the years and sites considered here. This exception is the result of an outlying observation that is approximately 50 times higher than the other observations in that time period. Benzene concentrations may also be higher in the winter months due to higher emissions or lower boundary layer (mixing level) heights. Only 0.4 percent of the total benzene measurements for all sites and years fall below the MDL in the AQS data files.

A spatial comparison of annual average benzene concentrations across the four sites is shown in Figure 3-8. Elizabeth consistently had the highest annual benzene concentrations of the four toxics sites followed by, in order of decreasing annual concentrations, Camden, New Brunswick, and Chester.

Figure 3-9, Figure 3-10, and Figure 3-11 contain the hourly average benzene concentrations measured at the PAMS sites of Camden, Rutgers University, and Rider University, respectively. These figures present both the average hourly concentrations for each day of the week (a) and the average of weekday and weekend concentrations (b). Only days with 24 hours of measurements were used in calculating the averages. While the exact peak and trough hours vary across the sites and across the days, the basic shape of the diurnal curves for benzene is similar for all sites. Peak benzene is in the early morning, the minimum is during the mid-afternoon, and concentrations start to rise again in the early evening. Emissions and reduction by the hydroxyl radical are major contributors to benzene's diurnal behavior, which may also be affected by a rising mixing height during the day. The peak is likely due to increased mobile emissions during the early morning commute. Benzene is destroyed by the hydroxyl radical, which is higher during daylight hours and substantially diminished at night. Fresh emissions may contribute to the rise of benzene concentrations after its early afternoon minimum.

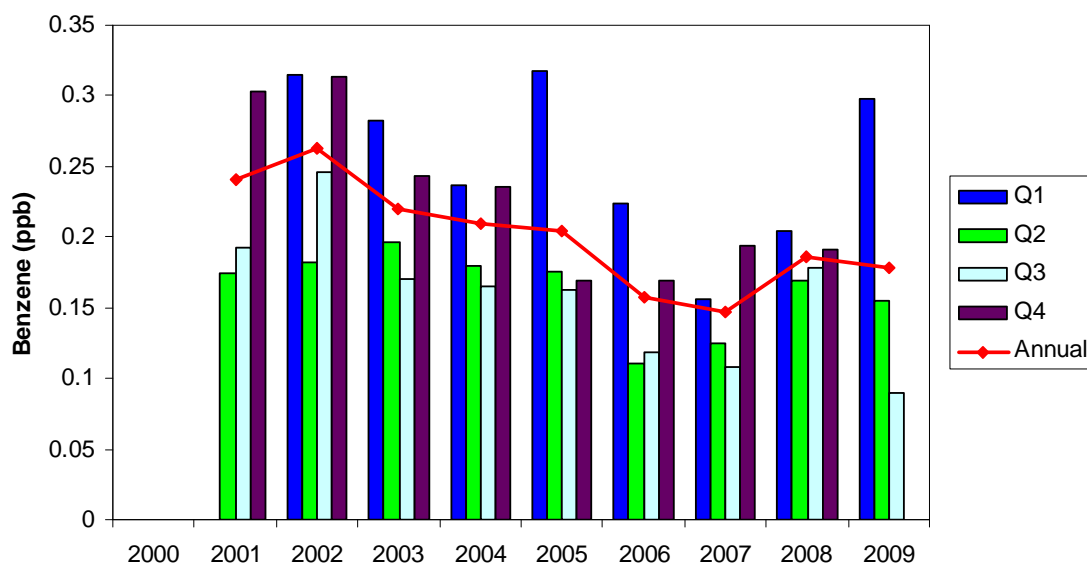
While the peak hour varies across the weekdays for Camden, it is clear that benzene peaks in the early morning and reaches a low in the early afternoon, when the hydroxyl radical concentrations and mixing height are highest. The average peak hour on a weekday is 5:00 AM, and the weekend peak is between 6:00 and 7:00 AM. There are likely not as many early morning emissions on the weekend. The minimum concentration timing and level is similar for weekdays and weekends and is around 2:00 or 3:00 PM at the Camden site. The weekday peaks and troughs are more uniform for Rutgers University. The average weekday sees a peak benzene concentration at 6:00 AM, and the concentrations are rather flat from 12:00 PM to 4:00 PM on average days. The minimum concentration is around 2:00 PM on the average weekday or 4:00 PM on the average weekend day. The Rider University site sees a peak around 6:00 and 7:00 AM for weekdays and weekends. The low is around 3:00 PM on the average weekday and 4:00 PM on the average weekend.

Figure 3-3. Quarterly and annual average benzene concentrations for 2000-2008 at Camden



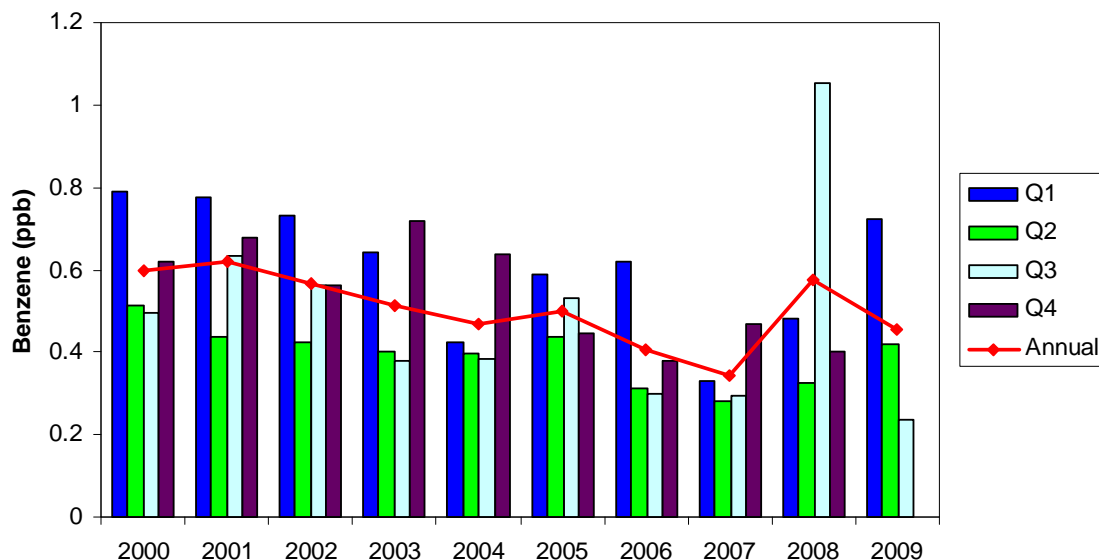
Note: Data were not available for 2009 or the last quarter of 2003 at Camden.

Figure 3-4. Quarterly and annual average benzene concentrations for 2001-2009 at Chester



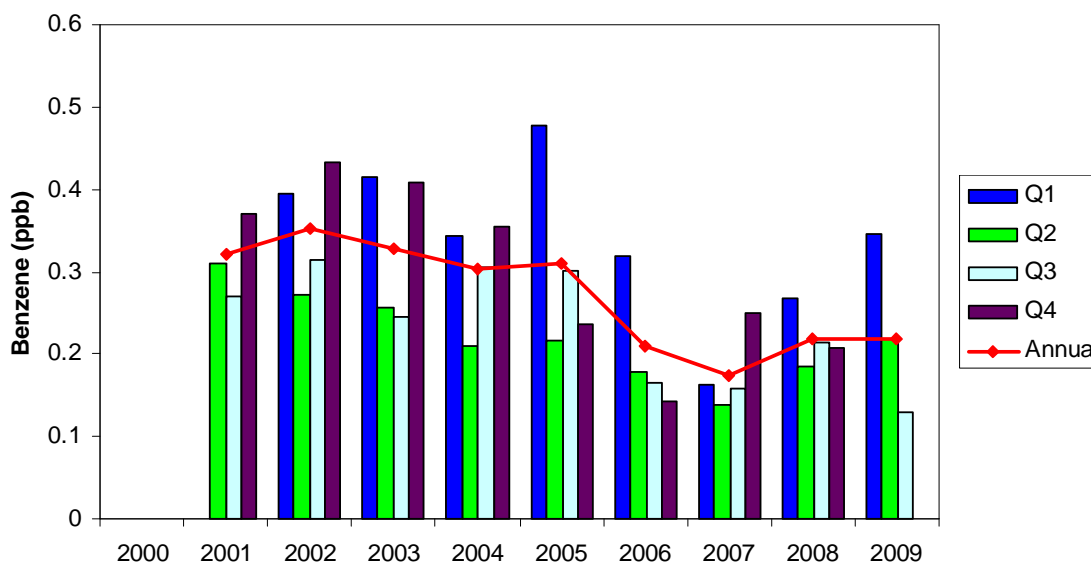
Note: At the time of retrieval, data were not available for the last quarter of 2009 at Chester. Chester came online in 2001.

Figure 3-5. Quarterly and annual average benzene concentrations for 2000-2009 at Elizabeth



Note: At the time of retrieval, data were not available for the last quarter of 2009 at Elizabeth.

Figure 3-6. Quarterly and annual average benzene concentrations for 2000-2009 at New Brunswick



Note: At the time of retrieval, data were not available for the last quarter of 2009 at New Brunswick.

Figure 3-7. Summer and winter average benzene concentrations at Camden, Chester, Elizabeth, and New Brunswick

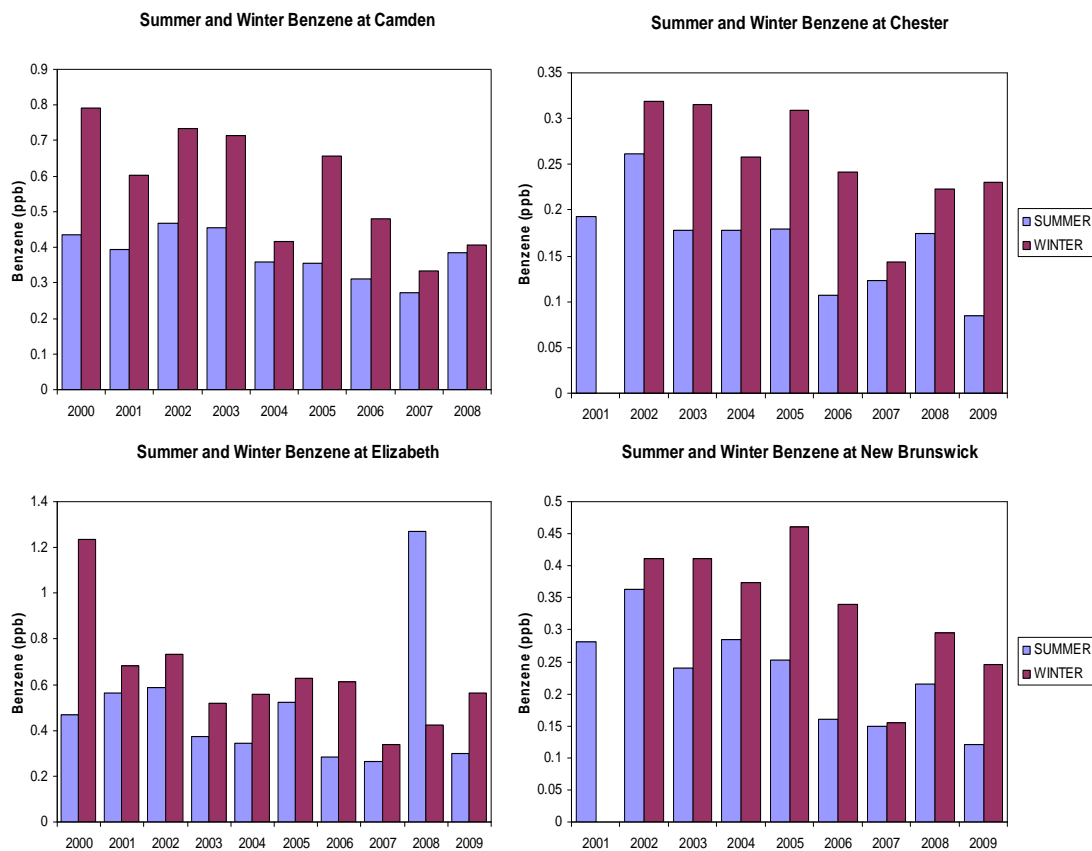


Figure 3-8. Comparison of annual average benzene concentrations by site

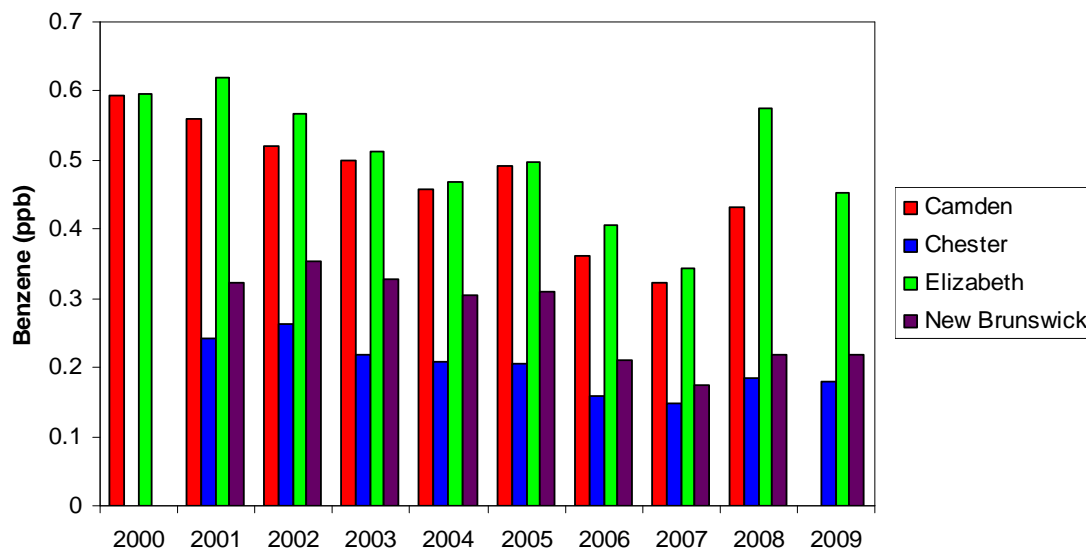


Figure 3-9. Comparison of average hourly benzene concentrations for 2000-2008 at the Camden PAMS

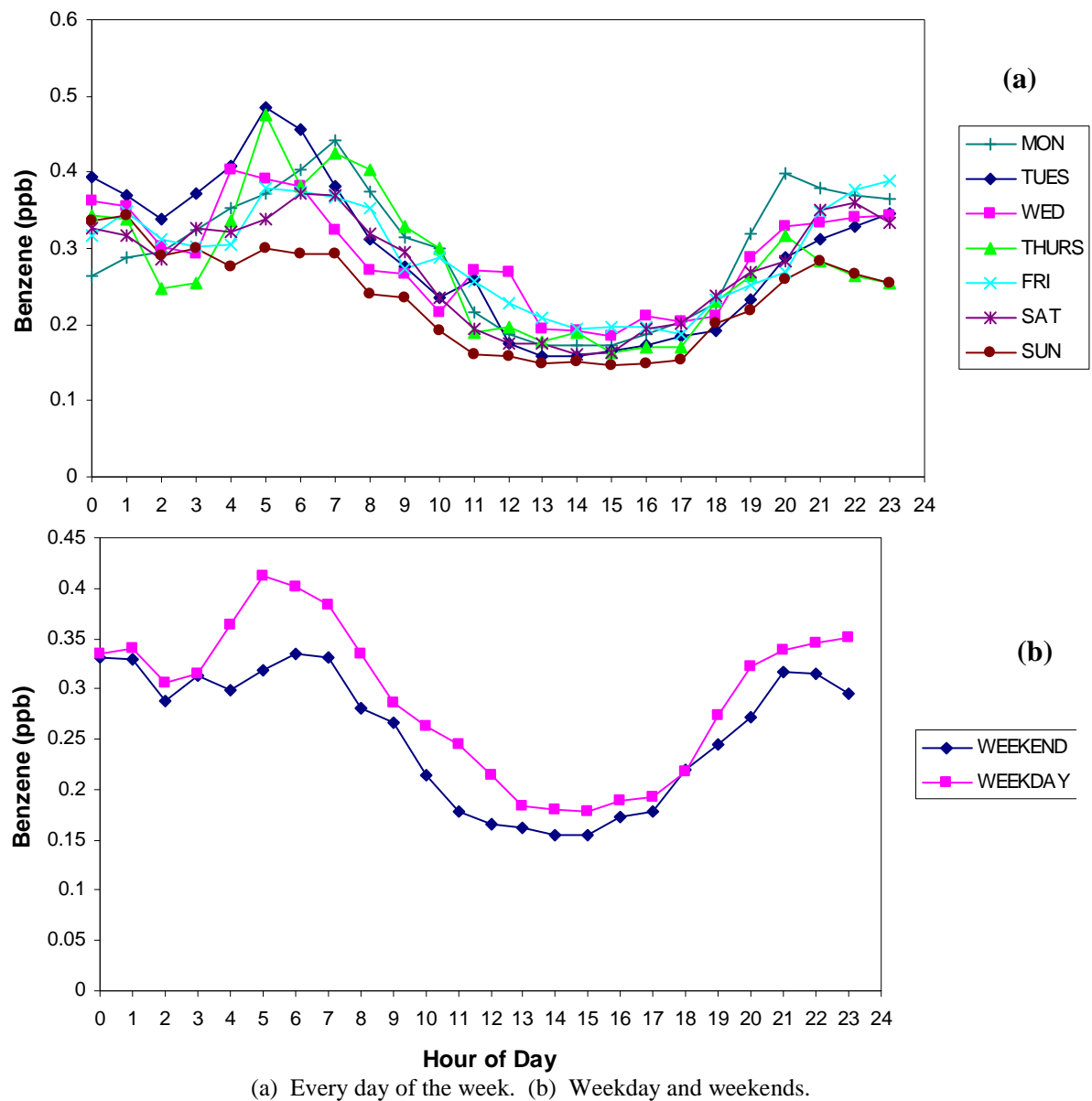
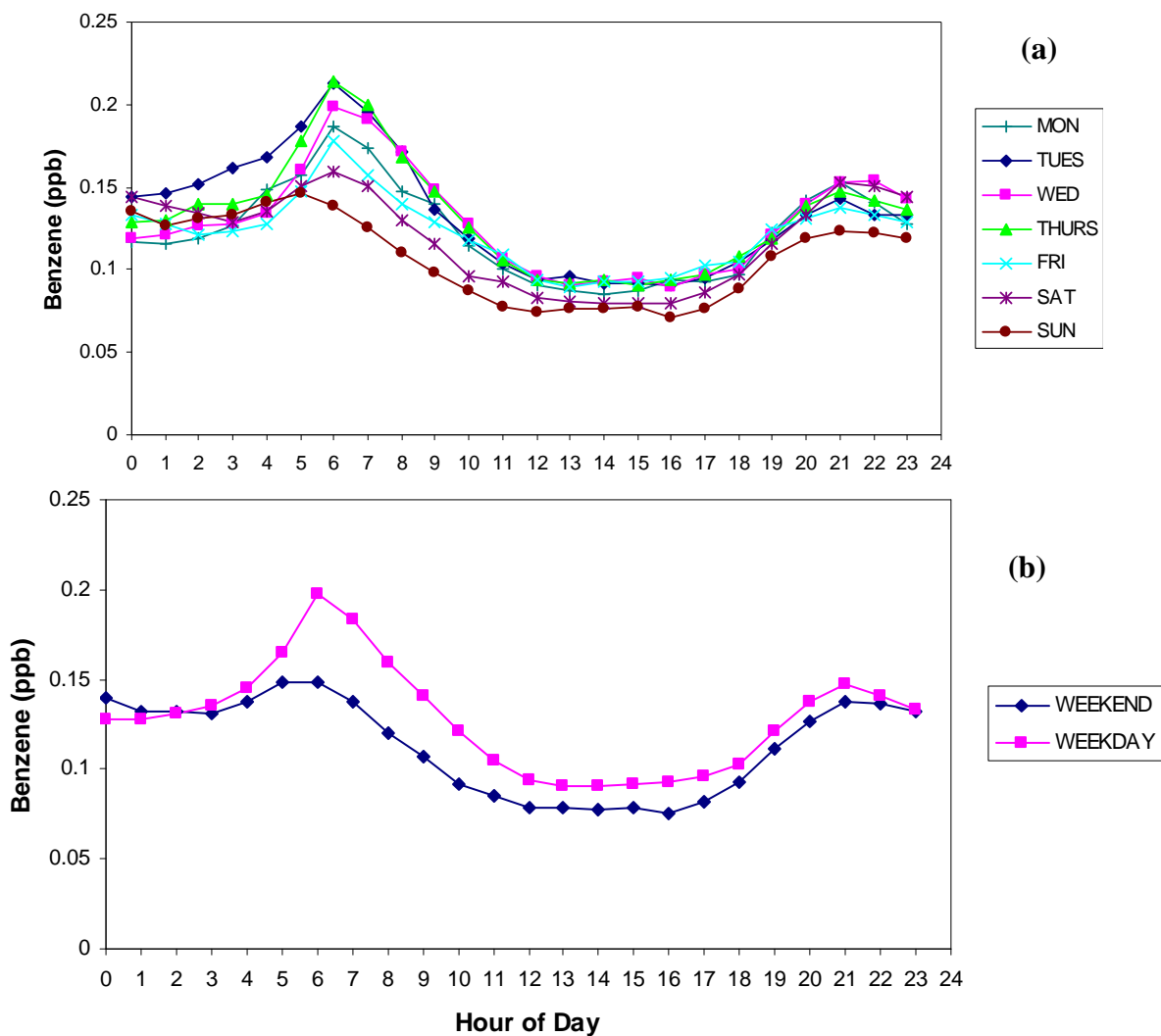
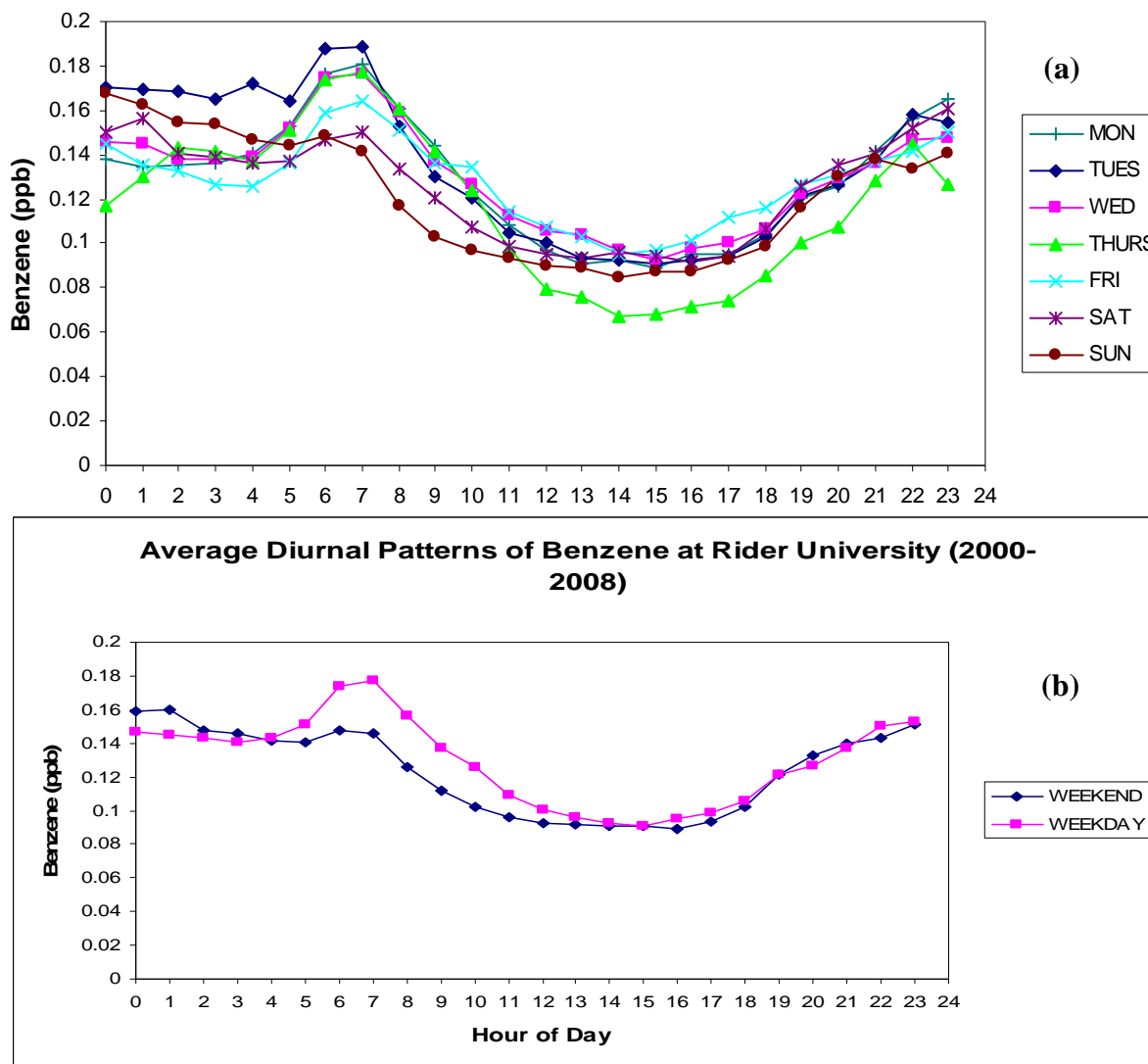


Figure 3-10. Comparison of average hourly benzene concentrations for 2000-2008 at the Rutgers University PAMS



(a) Every day of the week. (b) Weekday and weekends.

Figure 3-11. Comparison of average hourly benzene concentrations for 2000-2008 at the Rider University PAMS



(a) Every day of the week. (b) Weekday and weekends.

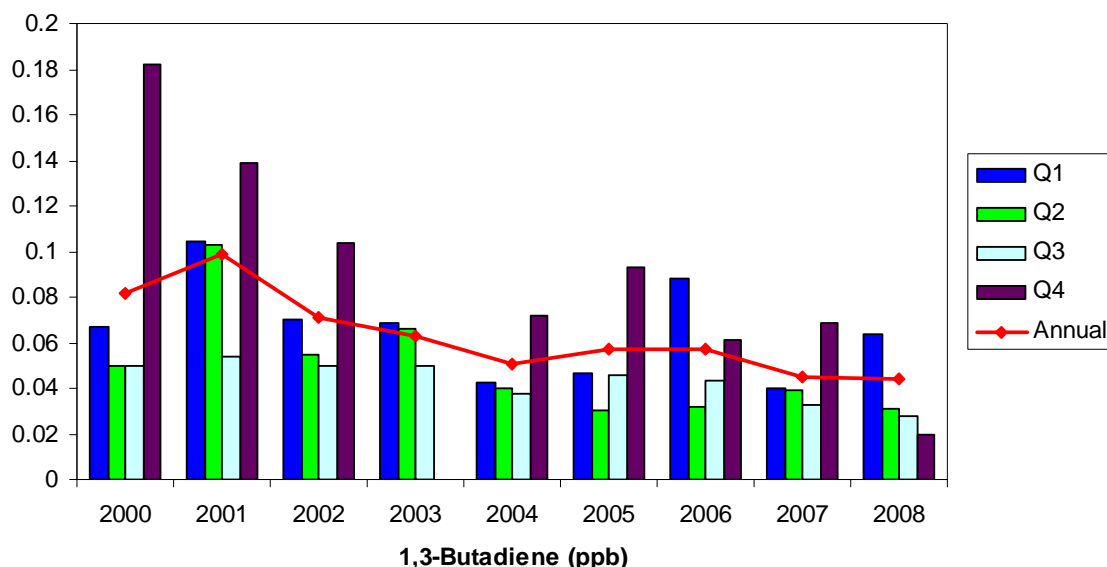
1,3-Butadiene

Figure 3-12 through Figure 3-15 contain the quarterly and annual average 1,3-butadiene concentrations for the four long-term New Jersey Air Toxics measurement sites. Of measured 1,3-butadiene concentrations, 54 percent are below the MDL, though the ratio has improved in later years. As a result, a large portion of the concentrations in these plots will be heavily weighted towards 50 percent of the MDL, especially in the early years. In some cases, what appeared to be a downward trend in measured concentrations was actually the result of a downward trend in reported MDL. At the Chester and New Brunswick sites, for example, it appeared that there was a clear downward trend between 2003 and 2004, but in reality, the MDL data from AQS decreased from 2003 to 2004. Because of the high percentage of points below the MDL, it is difficult to draw conclusions about 1,3-butadiene concentrations at these sites in the

early years. It is clear that the concentrations were low. One of the reasons for this is that 1,3-butadiene is readily broken down in the atmosphere to form other species (e.g., acrolein). Years 2006-2008 have markedly fewer observations below the reported MDLs. During these years, the first and fourth quarters (cooler months) had higher 1,3-butadiene concentrations than the second and third quarters (warmer months). The seasonal variation is even clearer in Figure 3-16. Winter concentrations were nearly always higher than summer average concentrations for a given year. This is a pattern typical of a species that is heavily influenced by photochemical degradation in the atmosphere.

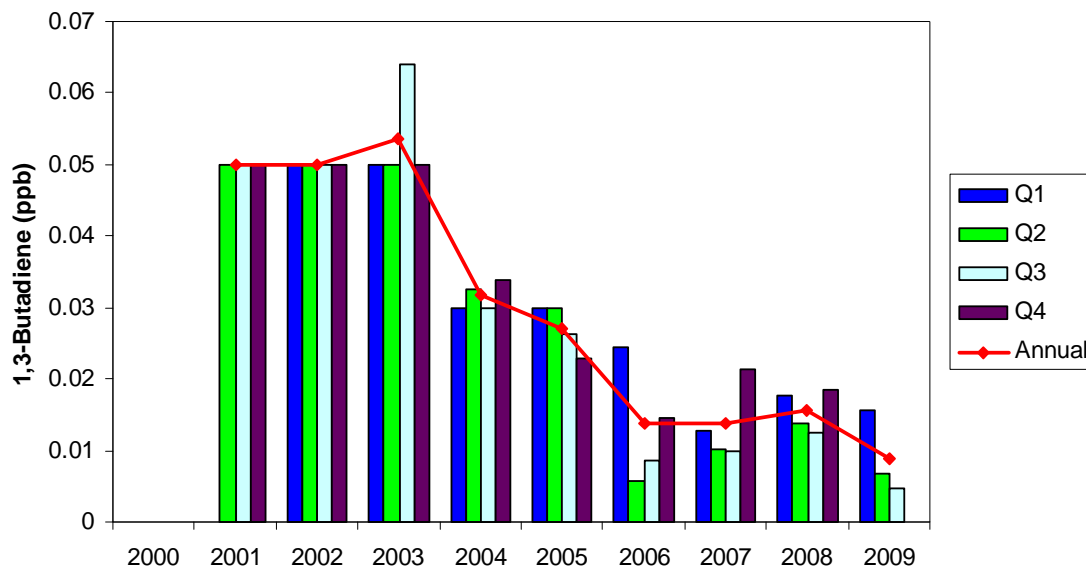
For all years, the relative concentration levels across the sites were similar. Elizabeth concentrations were highest, followed by Camden, then New Brunswick, and Chester. While the quarterly concentrations show the influence that photochemistry has on 1,3-butadiene concentrations, the consistent spatial distribution seen for each year in Figure 3-17 indicates that ambient 1,3-butadiene levels are also heavily influenced by the amount of local emissions as well.

Figure 3-12. Quarterly and annual average 1,3-butadiene concentrations for 2000-2008 at Camden



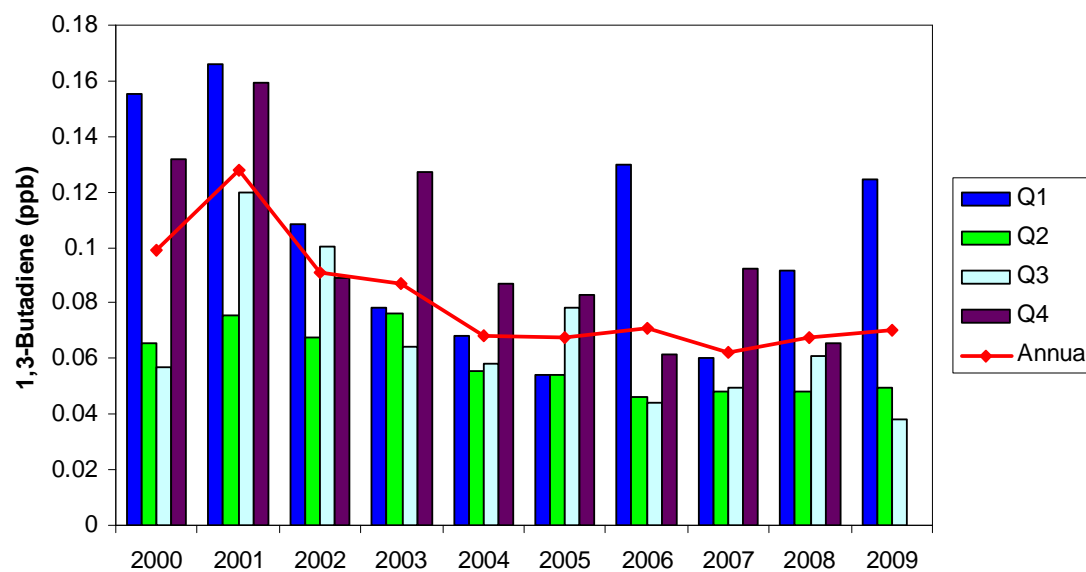
Note: Data were not available for 2009 or the last quarter of 2003 at Camden.

Figure 3-13. Quarterly and annual average 1,3-butadiene concentrations for 2001-2009 at Chester



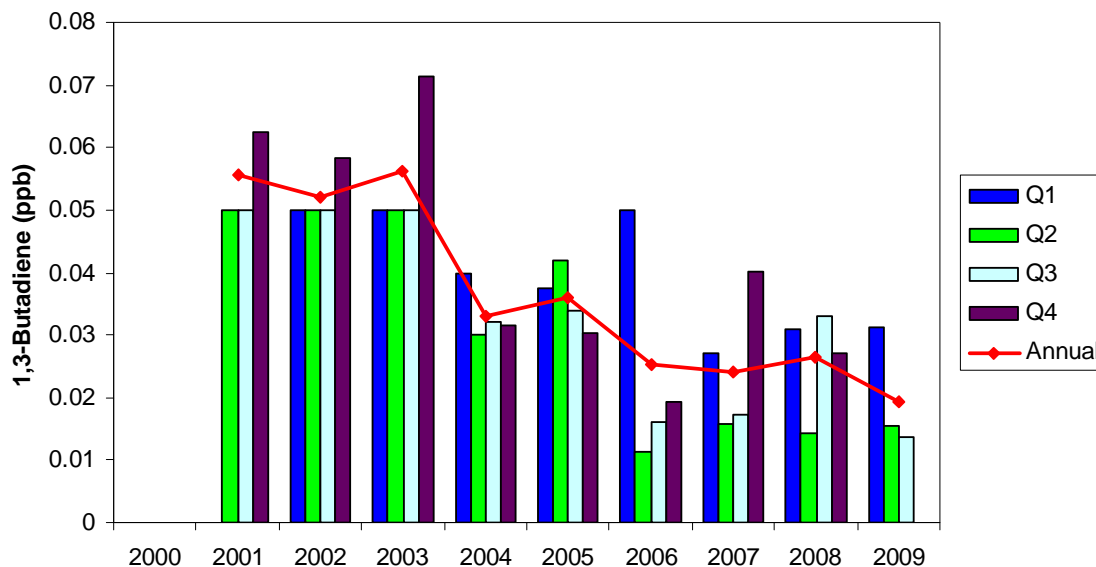
Note: At the time of retrieval, data were not available for the last quarter of 2009 at Chester. Chester came online in 2001.

Figure 3-14. Quarterly and annual average 1,3-butadiene concentrations for 2000-2009 at Elizabeth



Note: At the time of retrieval, data were not available for the last quarter of 2009 at Elizabeth.

Figure 3-15. Quarterly and annual average 1,3-butadiene concentrations for 2000-2009 at New Brunswick



Note: At the time of retrieval, data were not available for the last quarter of 2009 at New Brunswick.

Figure 3-16. Summer and winter average 1,3-butadiene concentrations at Camden, Chester, Elizabeth, and New Brunswick

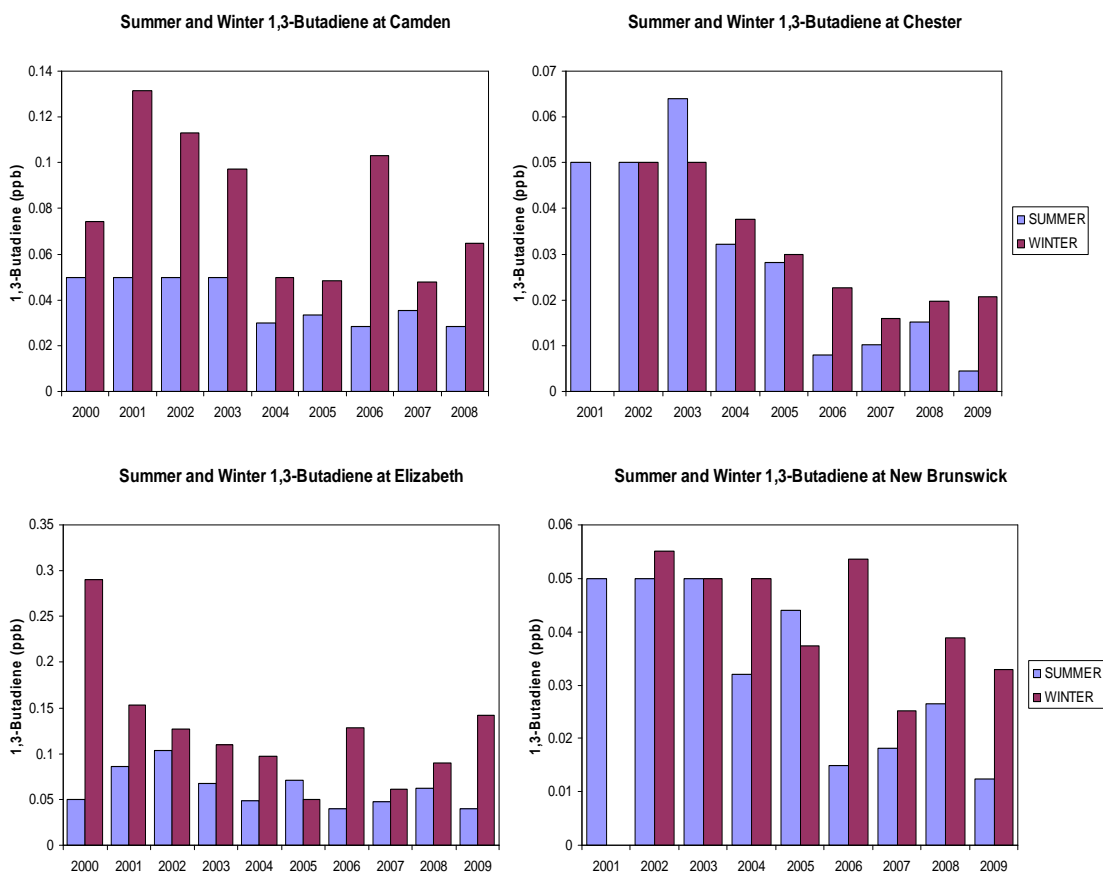
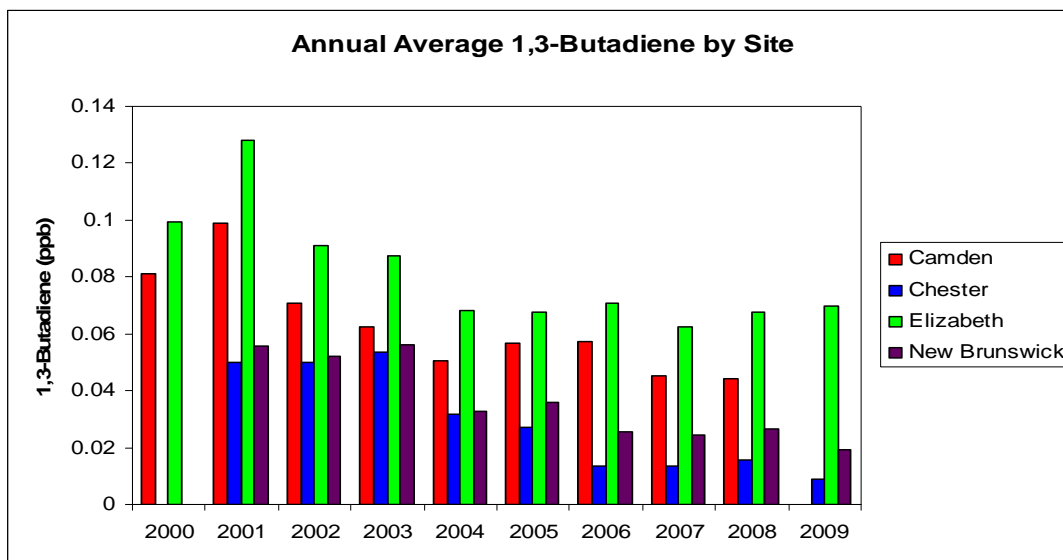


Figure 3-17. Comparison of annual average 1,3-butadiene concentrations by site

Xylenes

Xylene isomers are measured separately at each site. Figure 3-18 through Figure 3-21 show the quarterly and annual average *m,p*-xylene and *o*-xylene concentrations at the four long-term air toxics measurement sites in New Jersey. Only 2.4 percent of the *m,p*-xylene measurements are below the MDL, while 7.4 percent of the *o*-xylene measurements are. The measurements at Chester account for most of these low concentrations.

For all sites, there was an overall downward trend with concentration spikes in 2004 and 2008 at Elizabeth and slight jump in 2004 at Camden. The first quarter of 2001 at New Brunswick had very high average concentrations of xylenes, but this average was only based on six measurements. By 2003, the New Brunswick concentrations were at similar levels to the other sites.

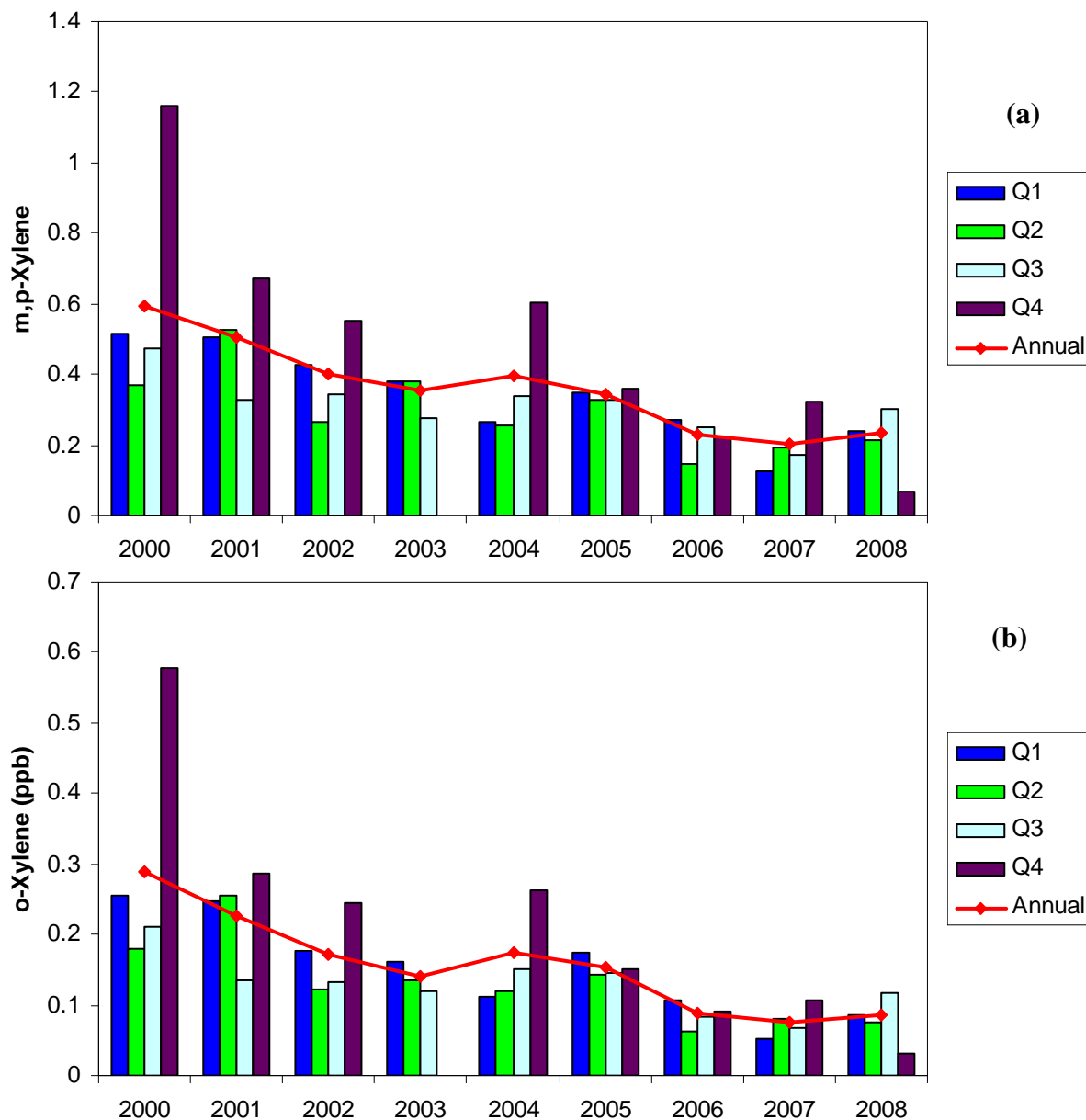
The quarterly behavior varied considerably between years and sites. For some years and sites, the concentrations in cooler quarters (1 and 4) were higher than warmer quarters (2 and 3), and in other years, the opposite was true. In Figure 3-22, Camden winter concentrations often exceeded summer concentrations. At Camden and at other sites, however, there were years when the summer average concentration exceeded that of the winter. There were also many years when winter and summer xylenes concentrations were very similar. This lack of a consistent quarterly trend may be related to the competing effects of emissions and chemistry or may be related to the more complex chemistry in which xylenes are involved (Seinfeld and Pandis, 1998).

In Figure 3-23, the annual average xylenes concentrations are compared for the four sites. While New Brunswick had the highest annual average total xylenes concentrations in earlier years, Elizabeth had the highest annual average concentrations

among the four sites in more recent years. Camden had the lowest concentrations in 2001 and 2002, and for the remaining years, Chester concentrations were the lowest.

Figure 3-24 through Figure 3-29 contain the hourly average *m,p*- and *o*-xylene isomer concentrations measured at the PAMS sites at Camden, Rutgers University, and Rider University, respectively. In the (a) plots, the average hourly concentrations for each day of the week are given. The (b) plots contain only the average weekday and weekend concentrations. Charts are separately presented for the *m,p*- and *o*-xylene isomers. Only days with 24 hours of measurements were used in calculating the averages. For most sites, the average peak in xylenes concentrations is between 5:00 and 6:00 AM. There is a deep trough in the afternoon, with xylenes concentrations reaching a minimum between 1:00 and 3:00 PM at all sites for weekdays and weekends. Weekend concentrations follow the same diurnal trend as weekdays though the morning peak is diminished and the concentrations tend to be lower. The early morning peak is likely due to morning mobile source emissions and the abrupt drop in concentrations can be attributed to the degradation of xylenes by the hydroxyl radical that is more prevalent during daylight hours, as well as a rising mixing height.

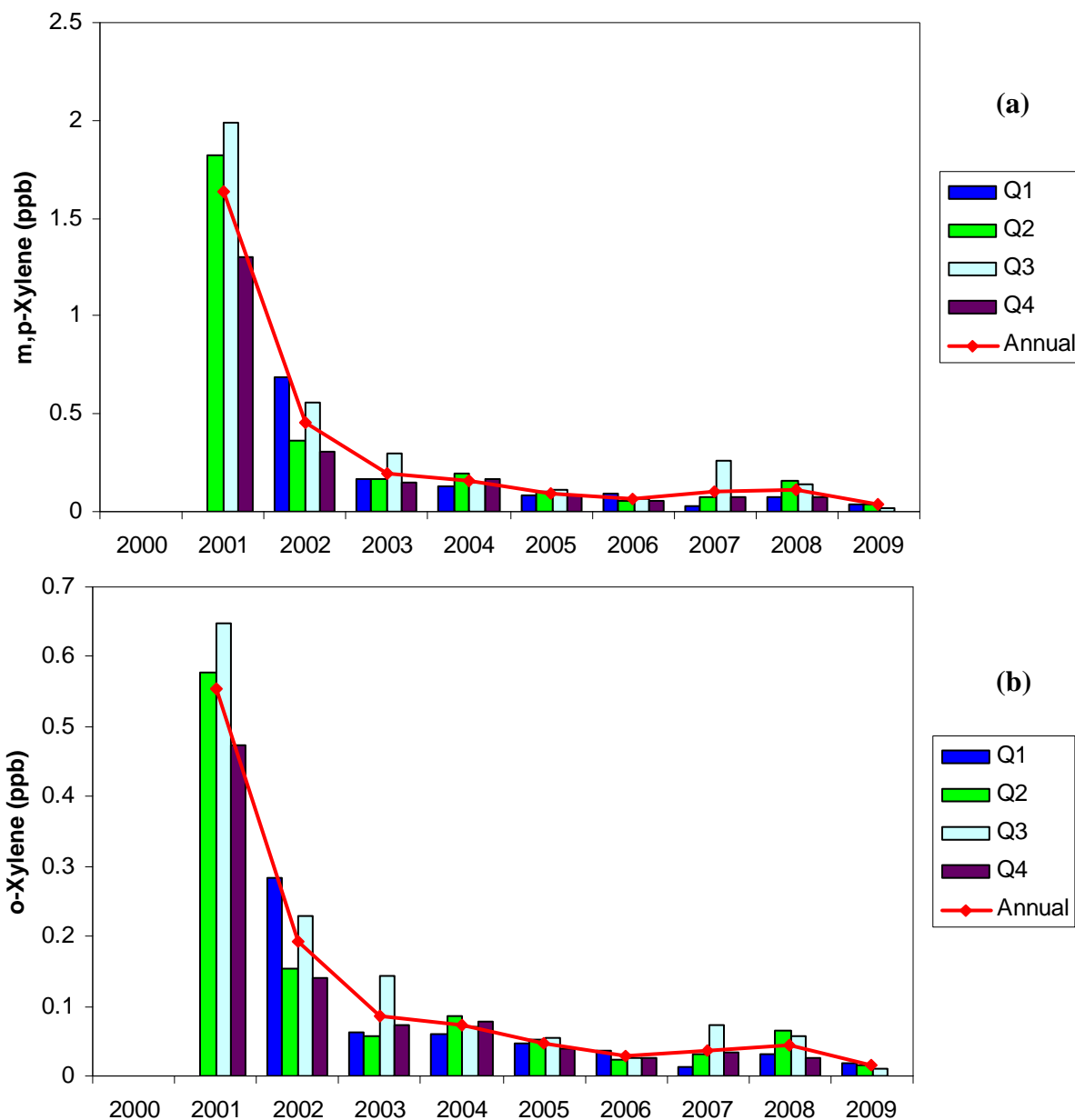
Figure 3-18. Quarterly and annual average xylenes concentrations for 2000-2008 at Camden



Note: Data were not available for 2009 or the last quarter of 2003 at Camden.

(a) m,p-Xylenes. (b) o-Xylene.

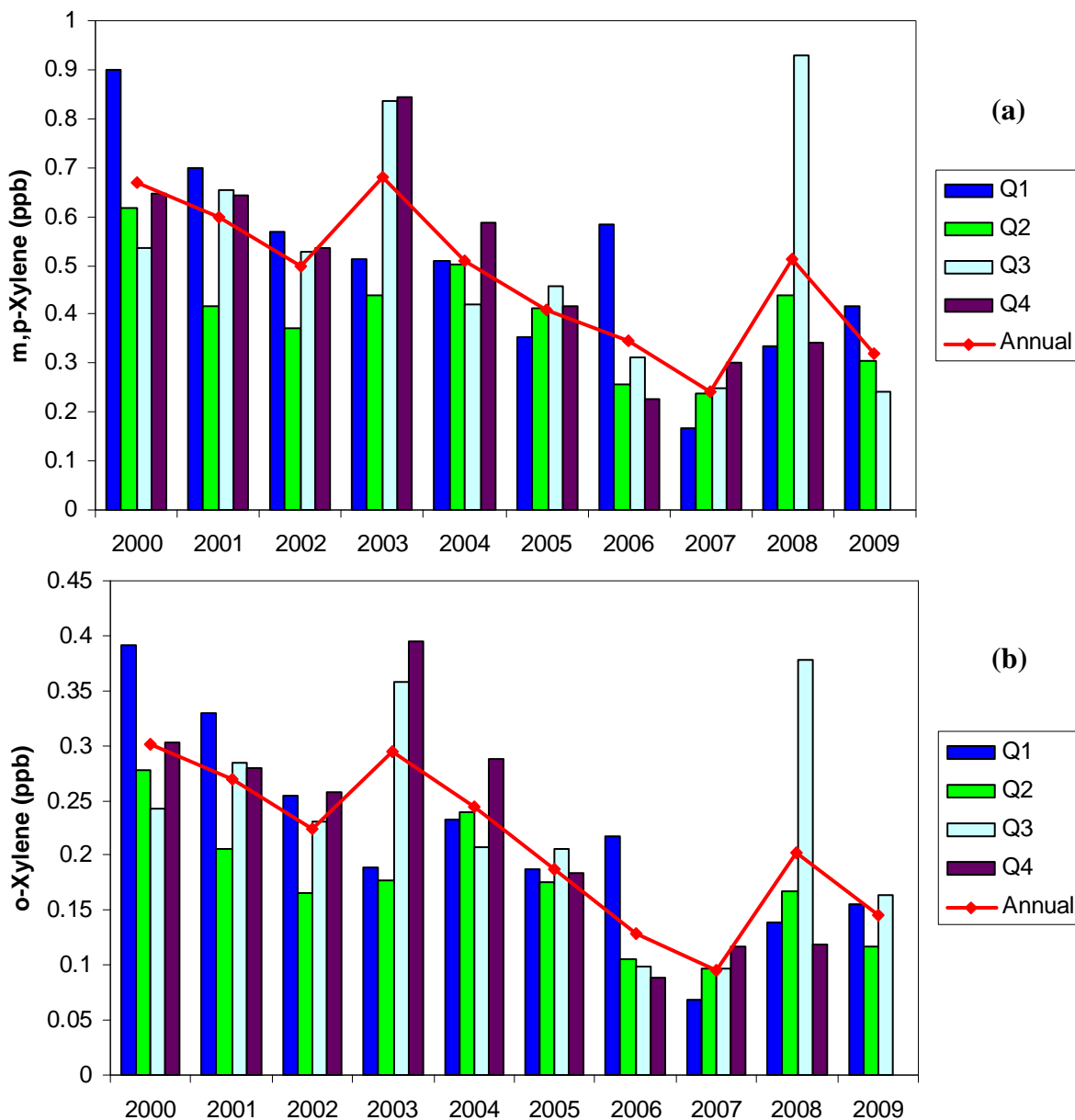
Figure 3-19. Quarterly and annual average xylenes concentrations for 2001-2009 at Chester



Note: At the time of retrieval, data were not available for the last quarter of 2009 at Chester. Chester came online in 2001.

(a) m,p-Xylenes. (b) o-Xylene.

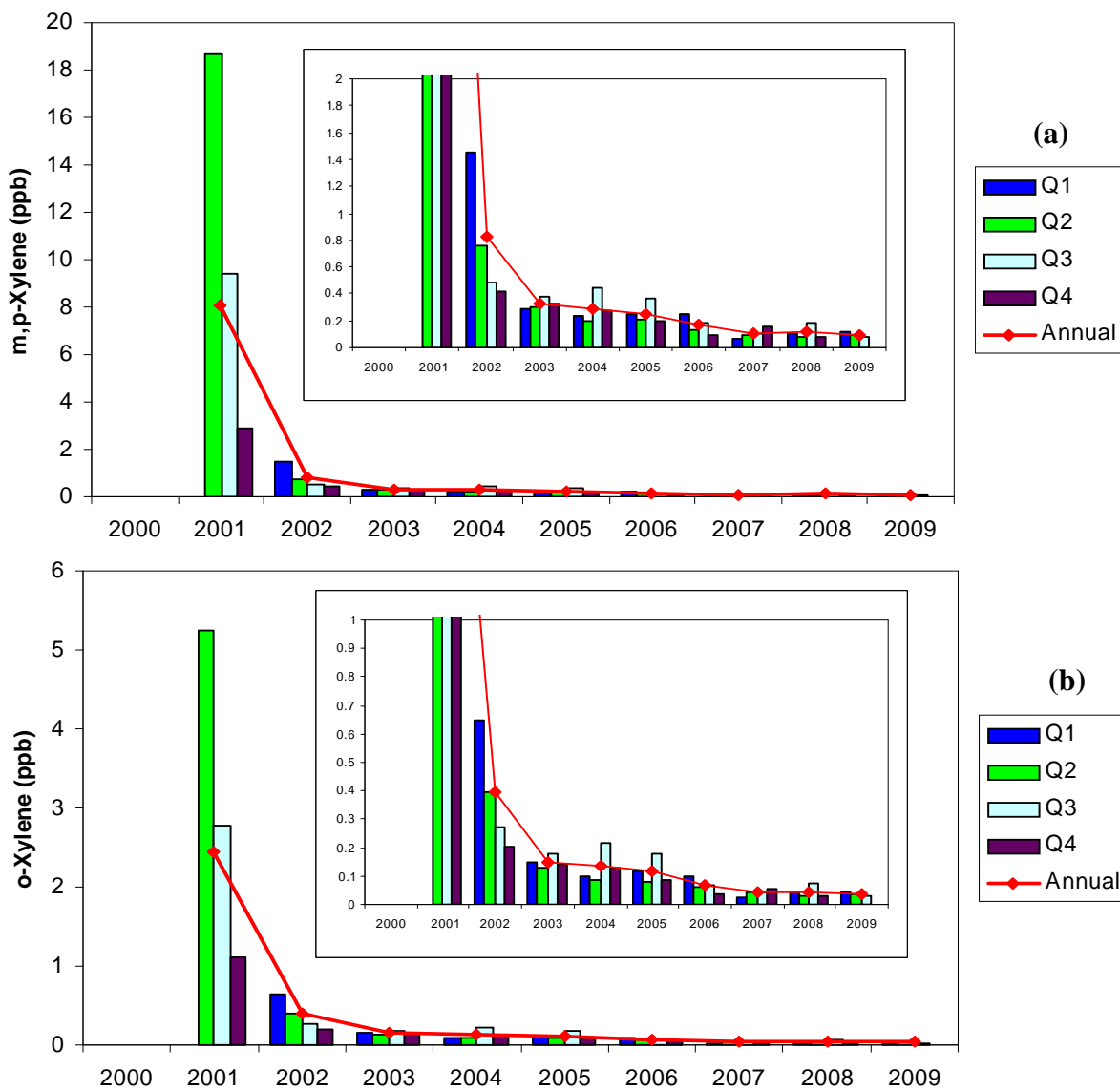
Figure 3-20. Quarterly and annual average xylenes concentrations for 2000-2009 at Elizabeth



Note: At the time of retrieval, data were not available for the last quarter of 2009 at Elizabeth.

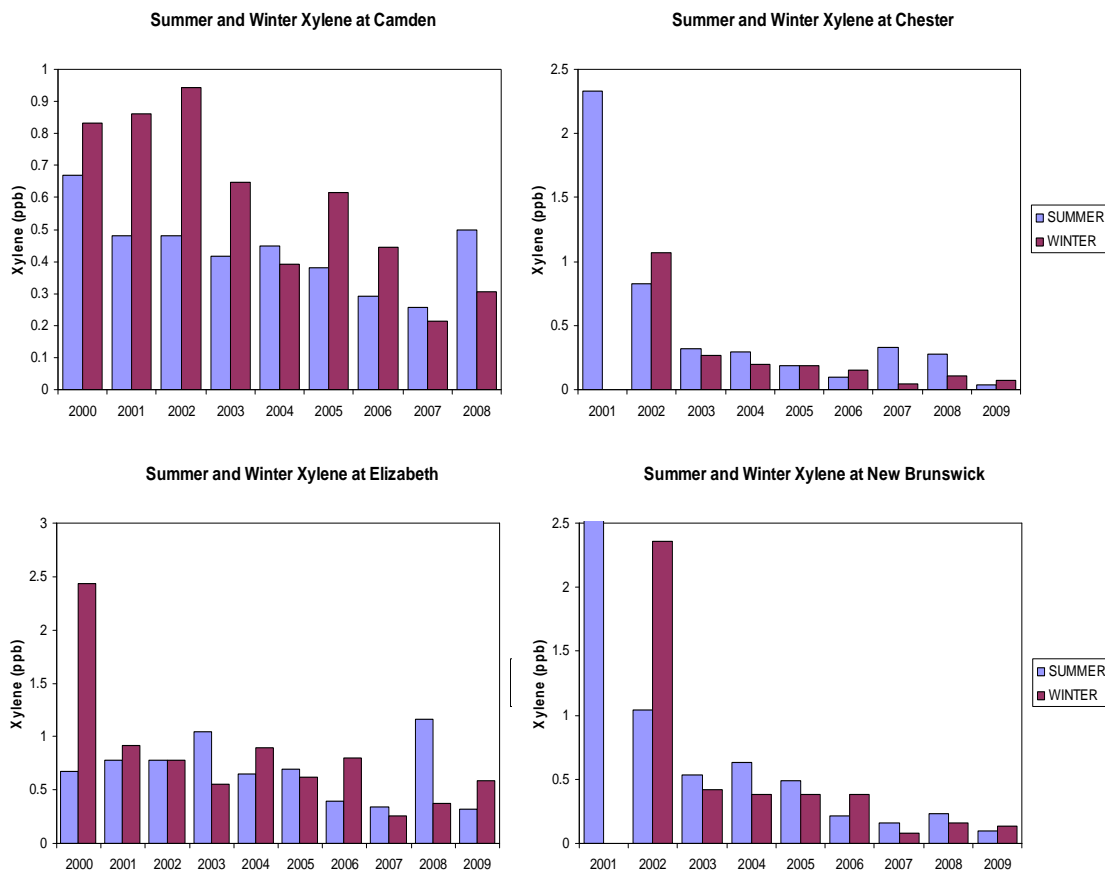
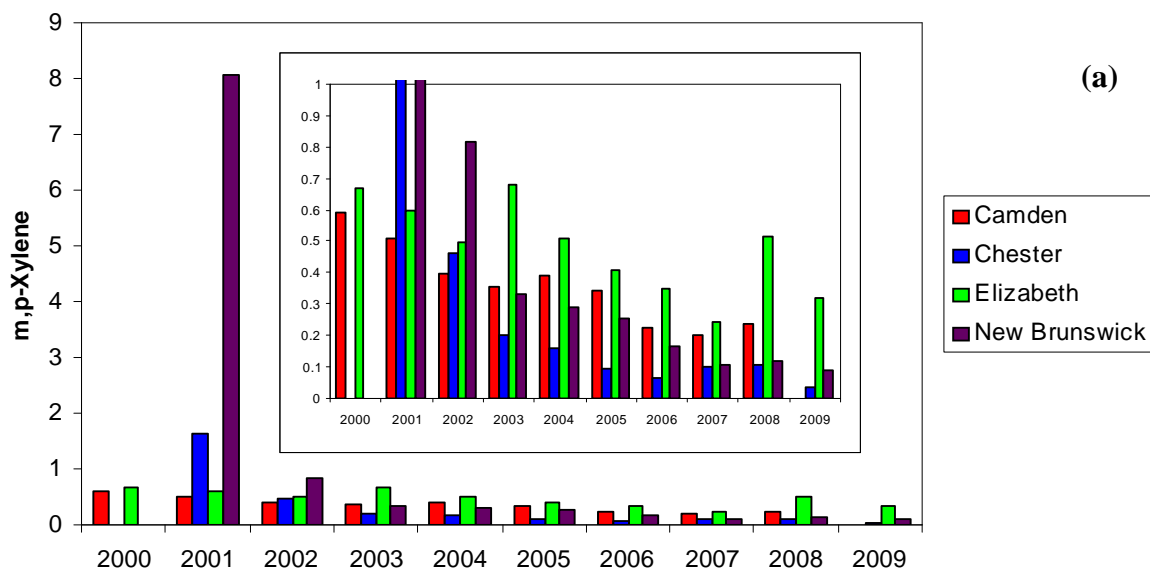
(a) m,p-Xylenes. (b) o-Xylene.

Figure 3-21. Quarterly and annual average xylenes concentrations for 2000-2009 at New Brunswick



Note: At the time of retrieval, data were not available for the last quarter of 2009 at New Brunswick.

(a) m,p-Xylenes. (b) o-Xylene.

Figure 3-22. Summer and winter average xylenes concentrations at Camden, Chester, Elizabeth, and New Brunswick**Figure 3-23. Comparison of annual average xylenes concentrations by site**

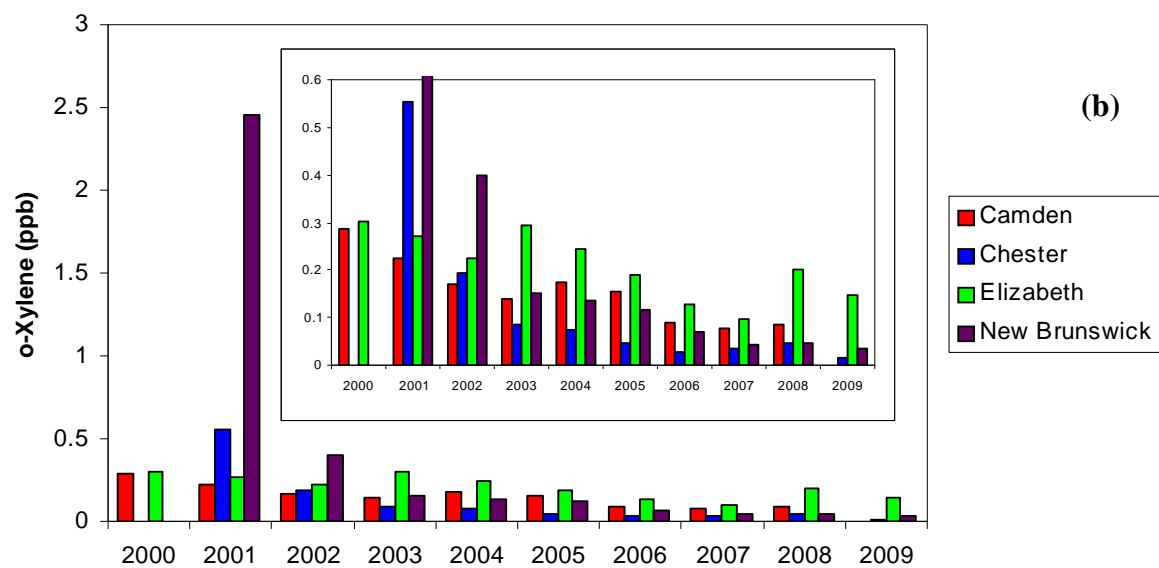
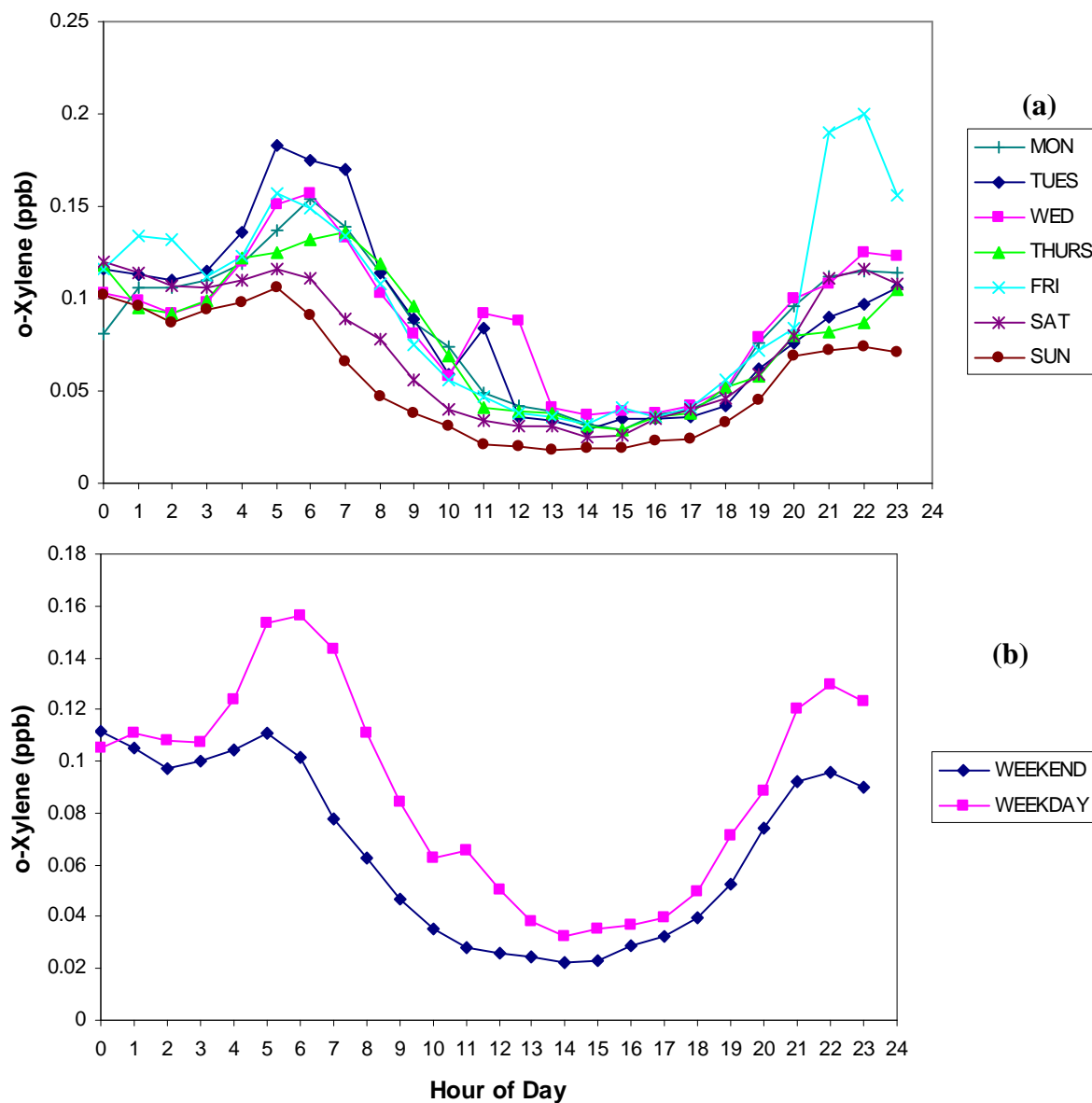


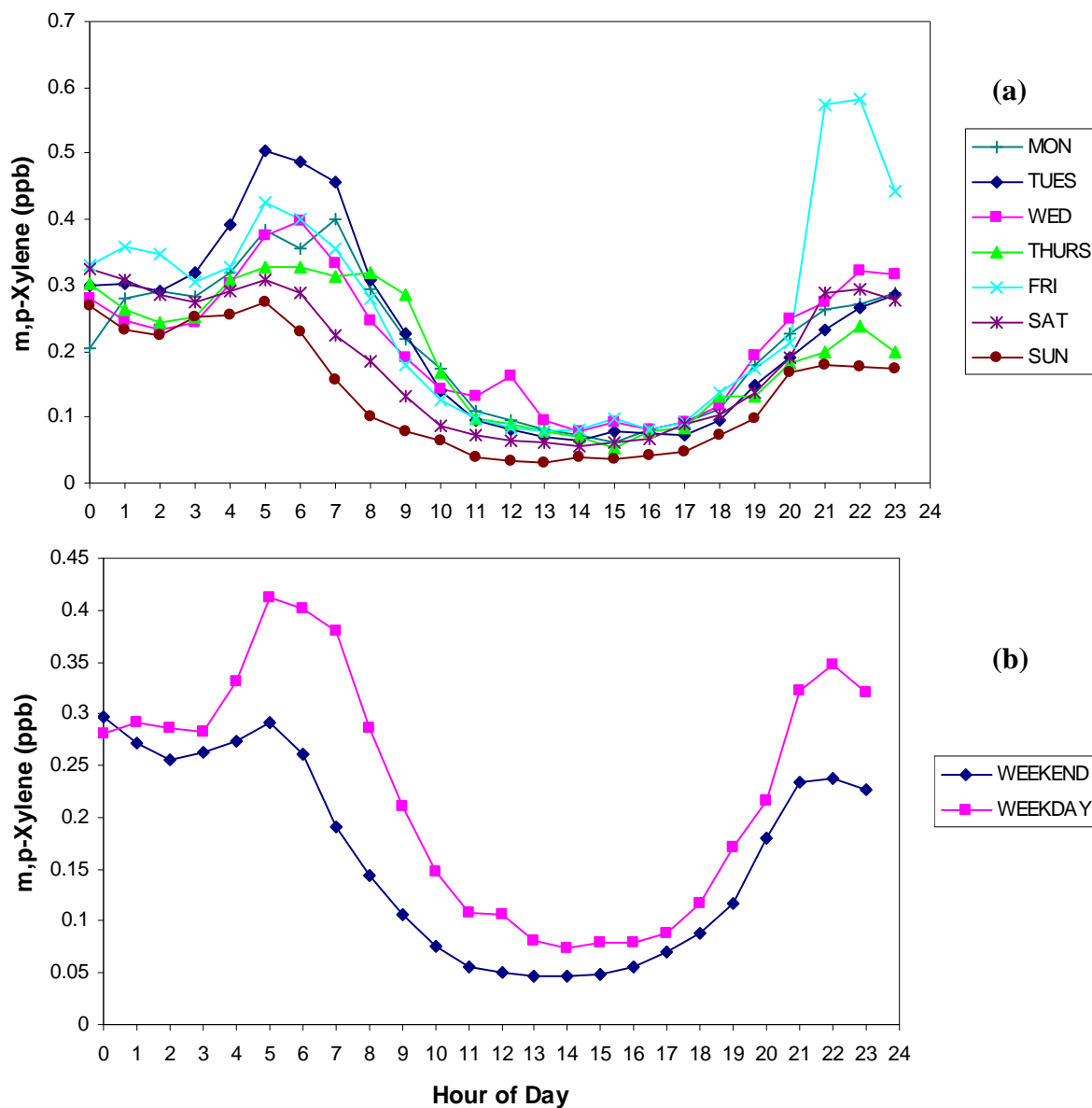
Figure 3-24. Comparison of average hourly *o*-xylene isomer concentrations for 2000-2008 at the Camden PAMS



(a) Every day of the week. (b) Weekday and weekends.

Note: The peak for hours 21-23 on Friday is due to several events where monitored concentrations were much higher than typical.

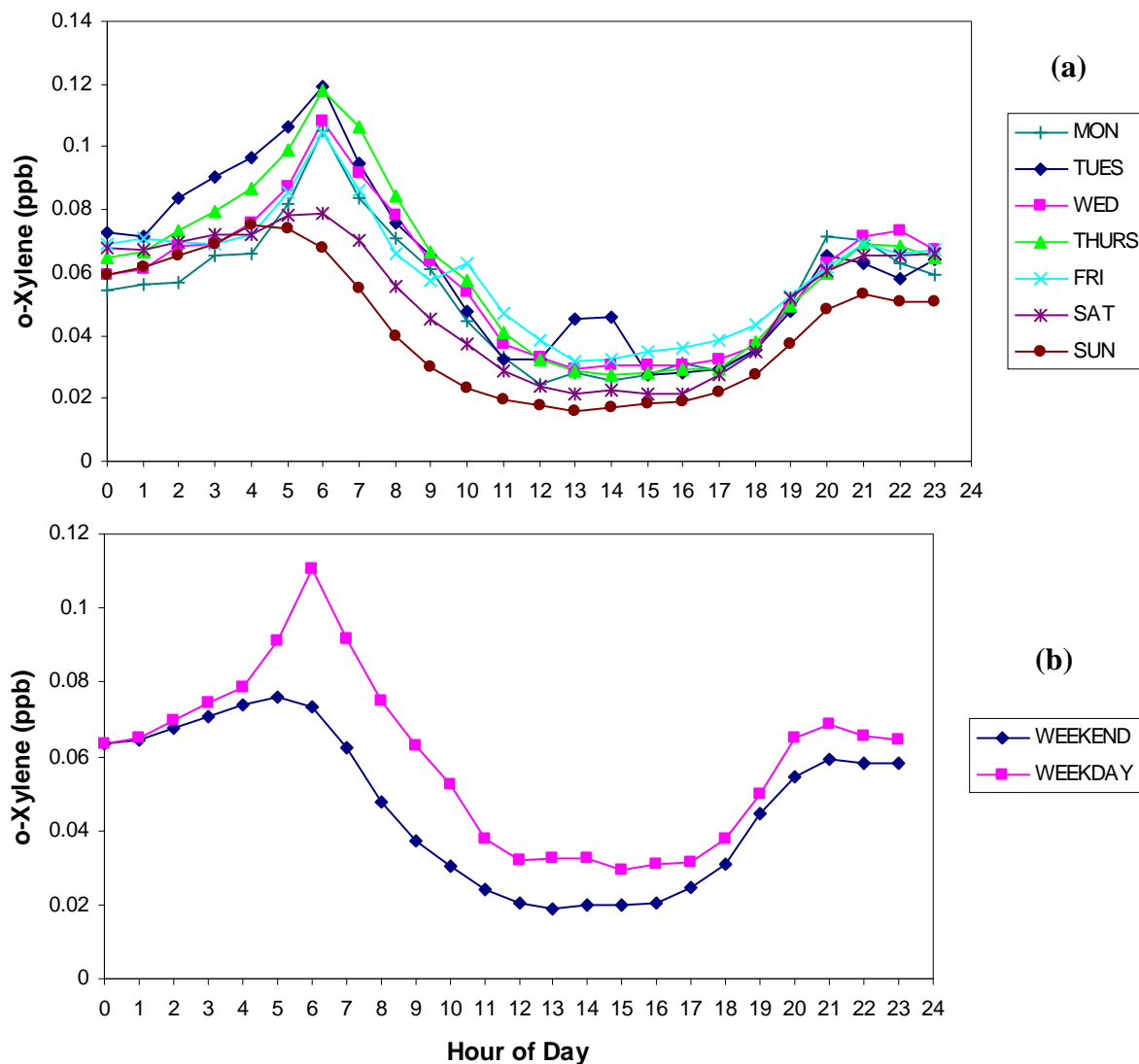
Figure 3-25. Comparison of average hourly *m,p*-xylene isomers concentrations for 2000-2008 at the Camden PAMS



(a) Every day of the week. (b) Weekday and weekends.

Note: The peak for hours 21-23 on Friday is due to several events where monitored concentrations were much higher than typical for those times.

Figure 3-26. Comparison of average hourly *o*-xylene isomer concentrations for 2000-2008 at the Rutgers University PAMS



(a) Every day of the week. (b) Weekday and weekends.

Figure 3-27. Comparison of average hourly *m,p*-xylene isomers concentrations for 2000-2008 at the Rutgers University PAMS

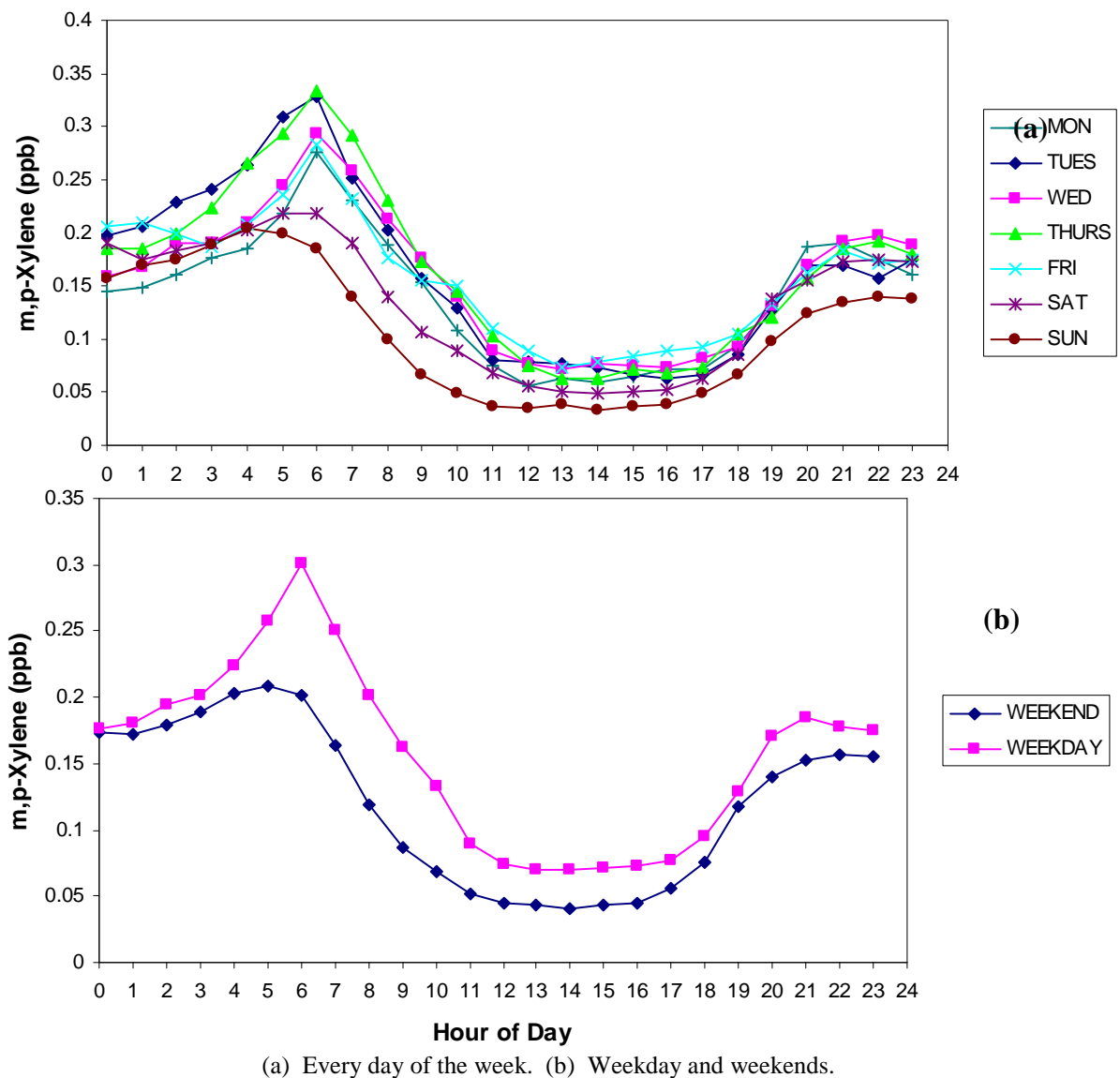
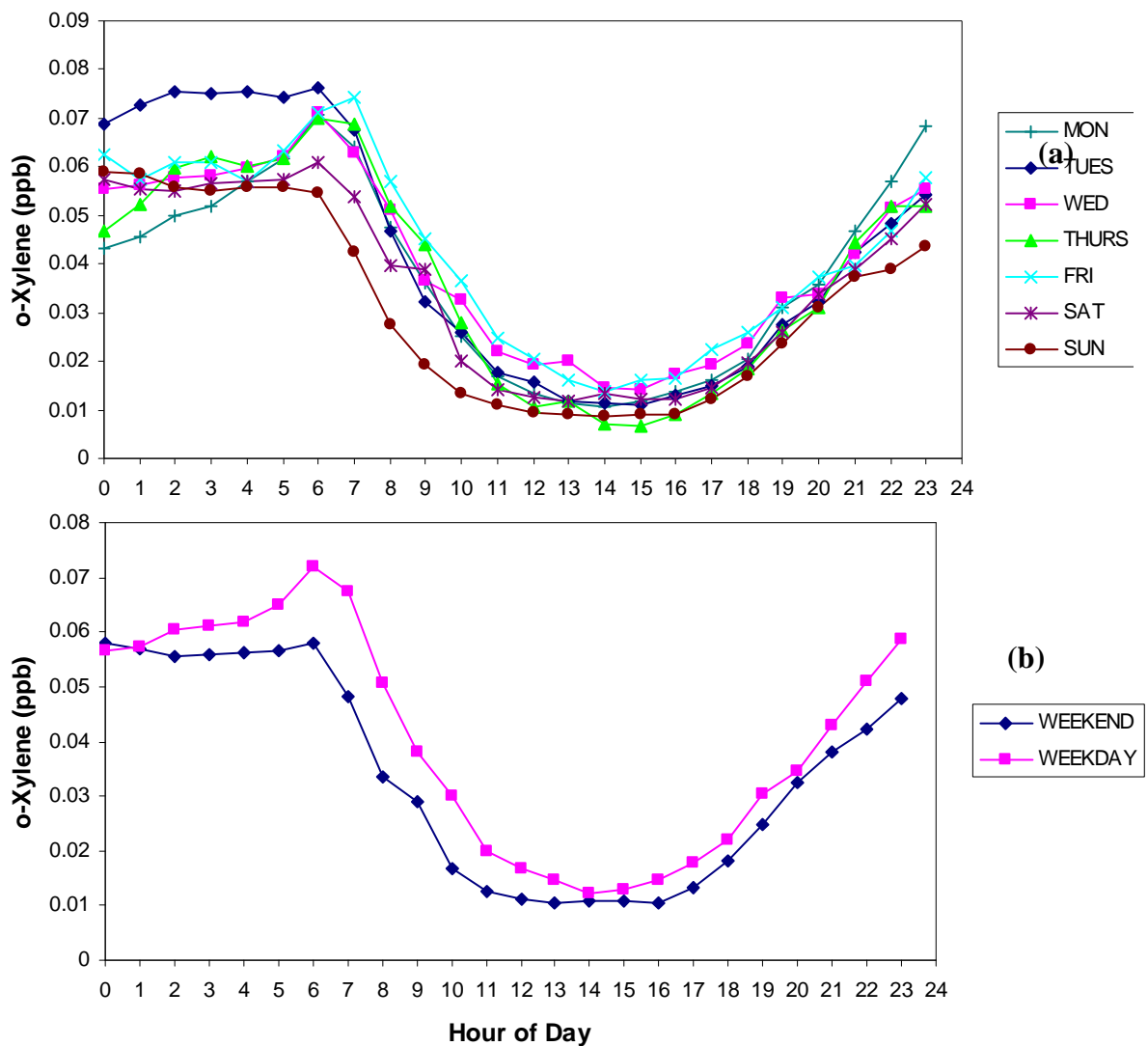
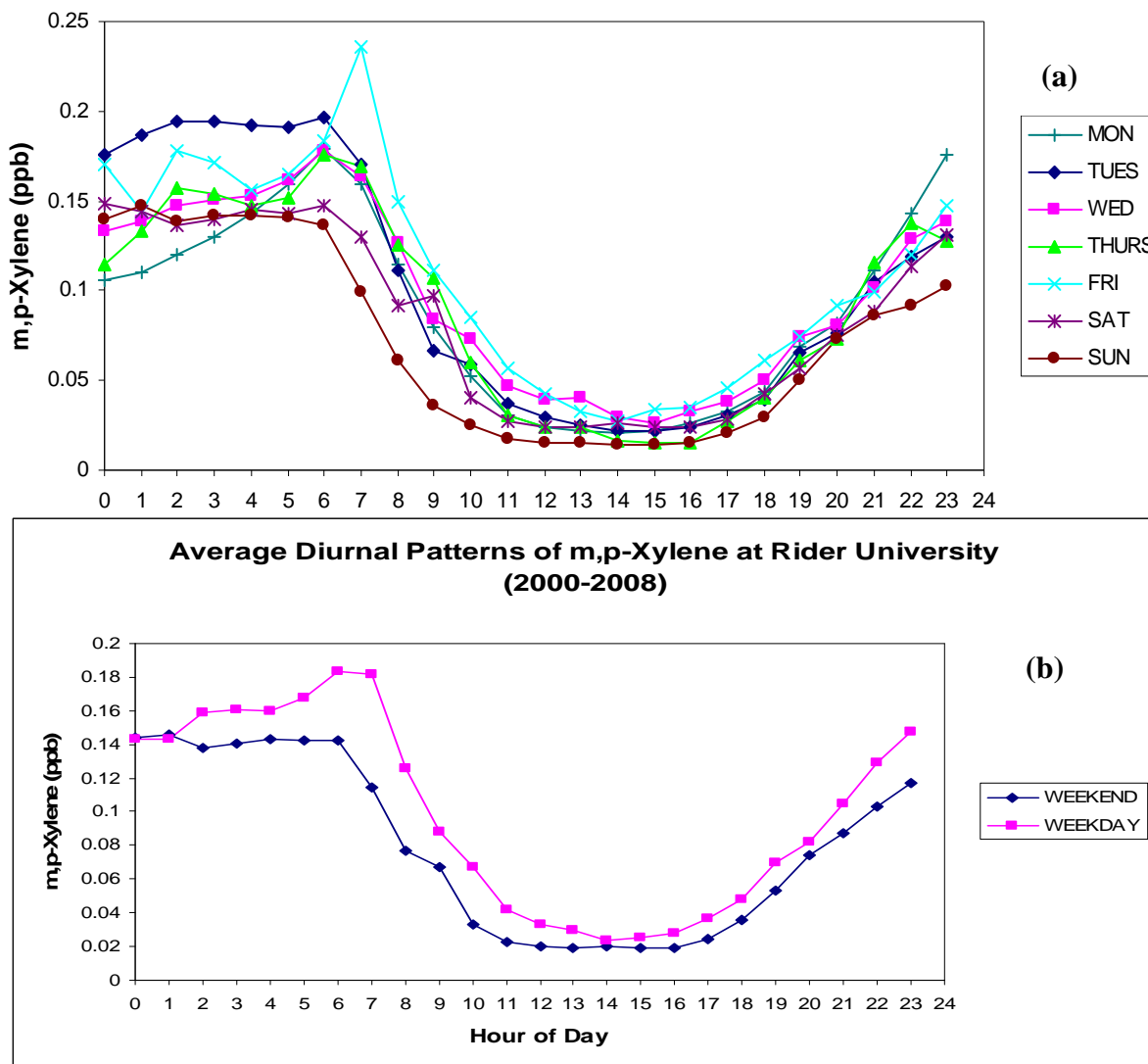


Figure 3-28. Comparison of average hourly *o*-xylene isomer concentrations for 2000-2008 at the Rider University PAMS



(a) Every day of the week. (b) Weekday and weekends.

Figure 3-29. Comparison of average hourly *m,p*-xylene isomers concentrations for 2000-2008 at the Rider University PAMS



(a) Every day of the week. (b) Weekday and weekends.

Toluene

Annual and quarterly concentrations for toluene are presented in Figure 3-30 through Figure 3-33 for the four NJ Air Toxics sites. Toluene concentrations followed a downward trend for the most part, with a jump in concentrations in 2008 at Camden and Elizabeth and a jump in 2007 at Chester. The quarterly behavior varied significantly between years and sites. Some of the extreme variations in average quarterly concentration are due to extreme outliers in the original dataset. For instance, a single 24-hour observation for toluene at Camden in the first quarter of 2008 was over 17 times the next highest observation in that time period, and approximately 50 times the median concentration for that time period. Conversely, the elevated levels of toluene during 2008 third quarter at Elizabeth and the second quarter at New Brunswick are the result of consistent measurement of high concentrations. While at Camden the average winter

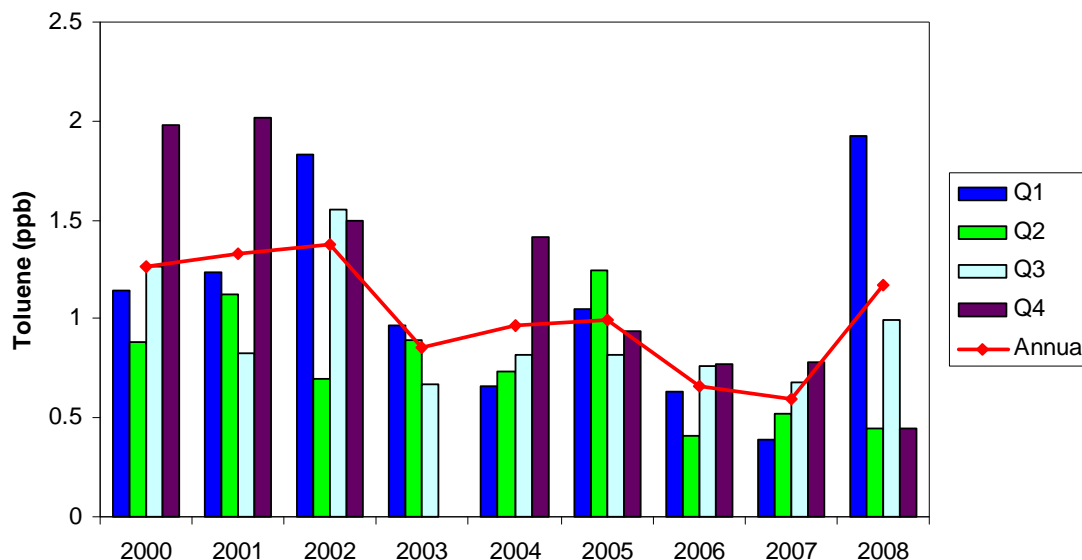
concentrations often exceeded the summer concentrations for a given year, there were many years and sites that saw similar summer and winter toluene concentrations (Figure 3-34). This lack of a consistent seasonal trend across all the sites might be due to the competing effects of emissions and chemistry or might reflect a more complex chemistry involving toluene compared to other species that are primarily influenced by direct/indirect photolysis only.

Only 0.1 percent of the toluene measurements were below the MDL for the years and sites considered here. Most of the sub-MDL concentrations were measured in earlier years.

Annual average toluene concentrations are compared across the four measurement sites in Figure 3-35. Camden and Elizabeth concentrations were similar for most years, though Elizabeth had the highest concentration of all sites from 2003 onwards. For most years, Chester saw the lowest annual toluene concentrations. There was more variability in “spatial trends” for toluene from year to year than for some other chemicals. For species like benzene, the relation between sites typically did not vary from year to year (e.g., Elizabeth > Camden > New Brunswick > Chester). While toluene concentrations usually followed a similar pattern, deviations were more common. New Brunswick provides a good example. New Brunswick saw the lowest concentration in 2007-2008 (instead of Chester) and the highest concentration in 2001-2002 (instead of Elizabeth).

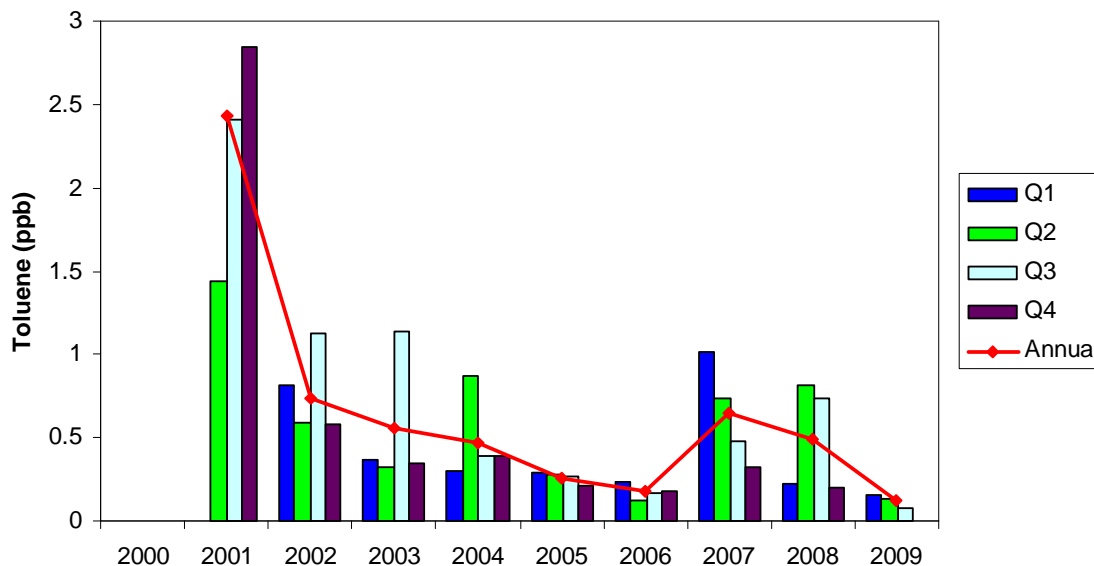
Figure 3-36 through Figure 3-38 contain the hourly average toluene concentrations measured at the PAMS sites at Camden, Rutgers University, and Rider University. In the (a) plots, the average hourly concentrations for each day of the week are given. The (b) plots show only average weekday and weekend concentrations. Only days with 24 hours of measurements were used in calculating the averages. For most sites, the average peak in toluene concentrations is around 5:00 or 6:00 AM, but at Rider University and Camden, the average weekend values do not peak very much. Toluene levels go down at all sites over the course of the day, reaching a minimum in the early afternoon. Concentrations rise again starting in the early evening, as the sun goes down. Weekend concentrations follow the same diurnal trends as weekdays, but weekend concentrations are lower from about 5:00 or 6:00 AM onwards. The early morning peak is likely due to morning mobile source emissions and the afternoon trough is likely due to photochemical reactions that consume toluene in the atmosphere as well as a higher mixing height.

Figure 3-30. Quarterly and annual average toluene concentrations for 2000-2008 at Camden



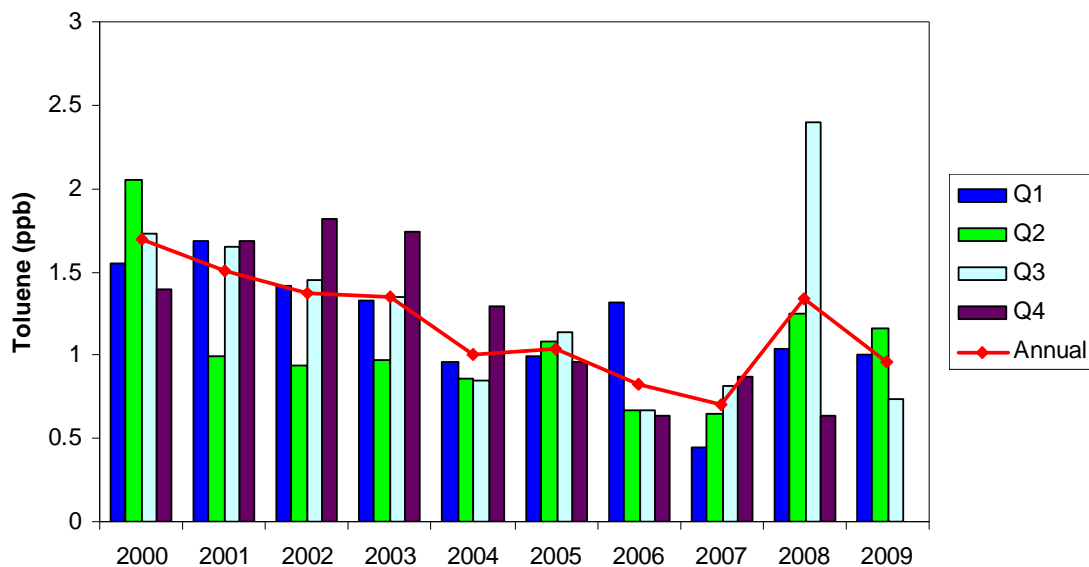
Note: Data were not available for 2009 or the last quarter of 2003 at Camden.

Figure 3-31. Quarterly and annual average toluene concentrations for 2001-2009 at Chester



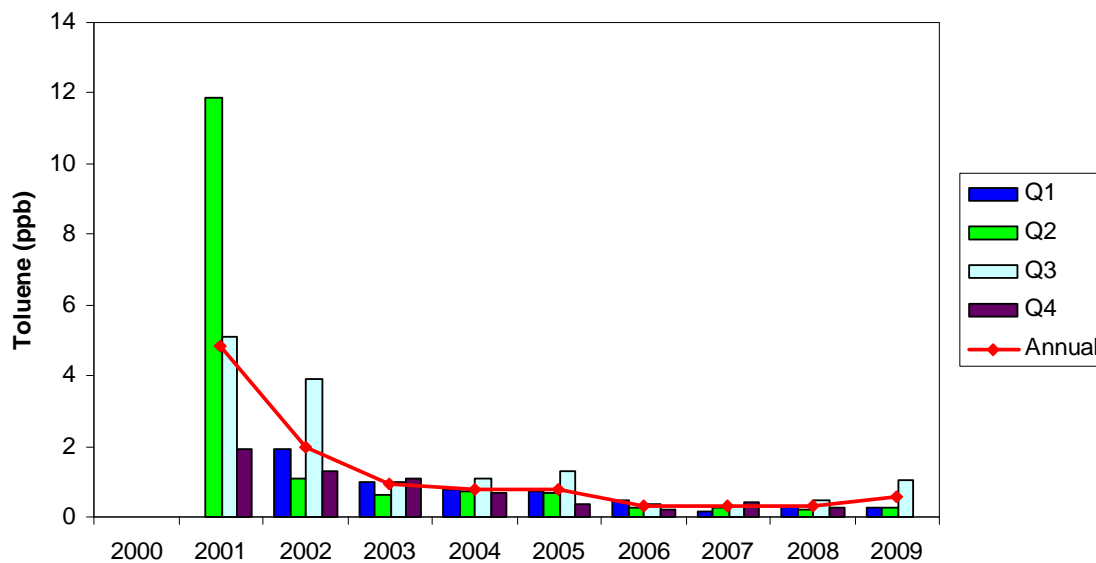
Note: At the time of retrieval, data were not available for the last quarter of 2009 at Chester. Chester came online in 2001.

Figure 3-32. Quarterly and annual average toluene concentrations for 2000-2009 at Elizabeth



Note: At the time of retrieval, data were not available for the last quarter of 2009 at Elizabeth.

Figure 3-33. Quarterly and annual average toluene concentrations for 2000-2009 at New Brunswick



Note: At the time of retrieval, data were not available for the last quarter of 2009 at New Brunswick.

Figure 3-34. Summer and winter average toluene concentrations at Camden, Chester, Elizabeth, and New Brunswick

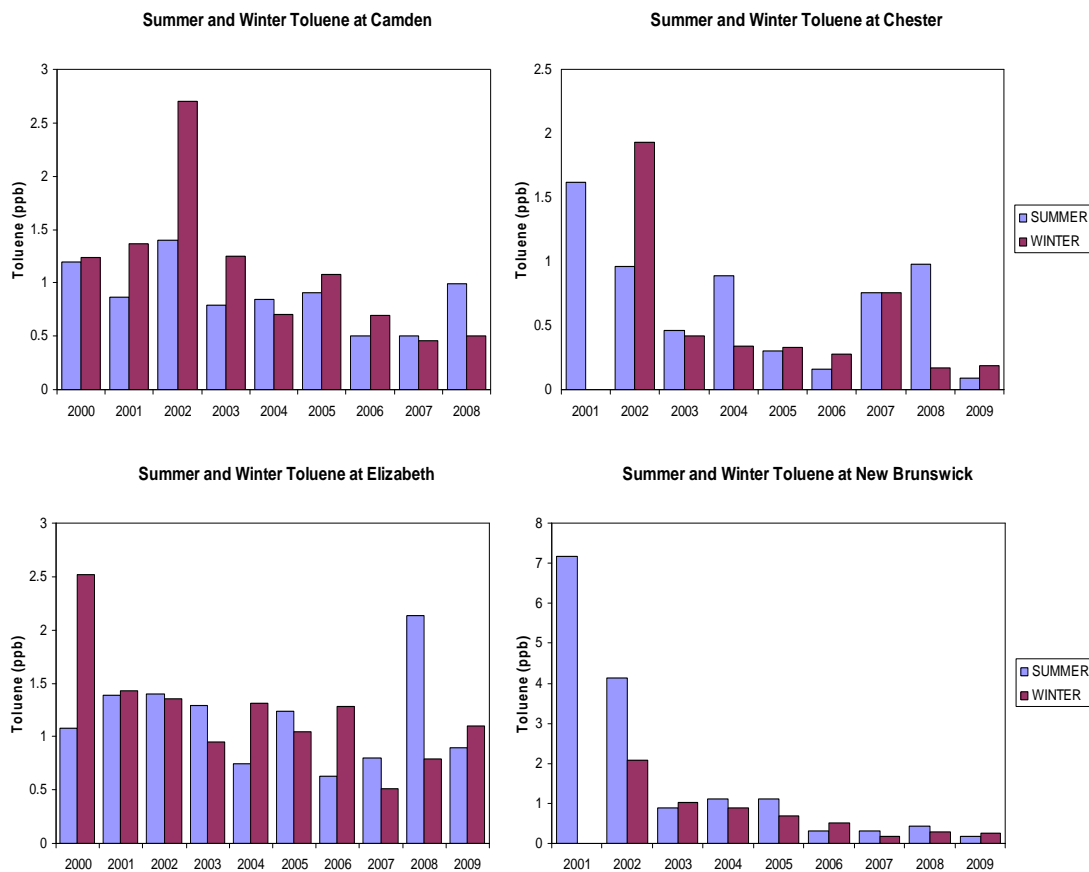


Figure 3-35. Comparison of annual average toluene concentrations by site

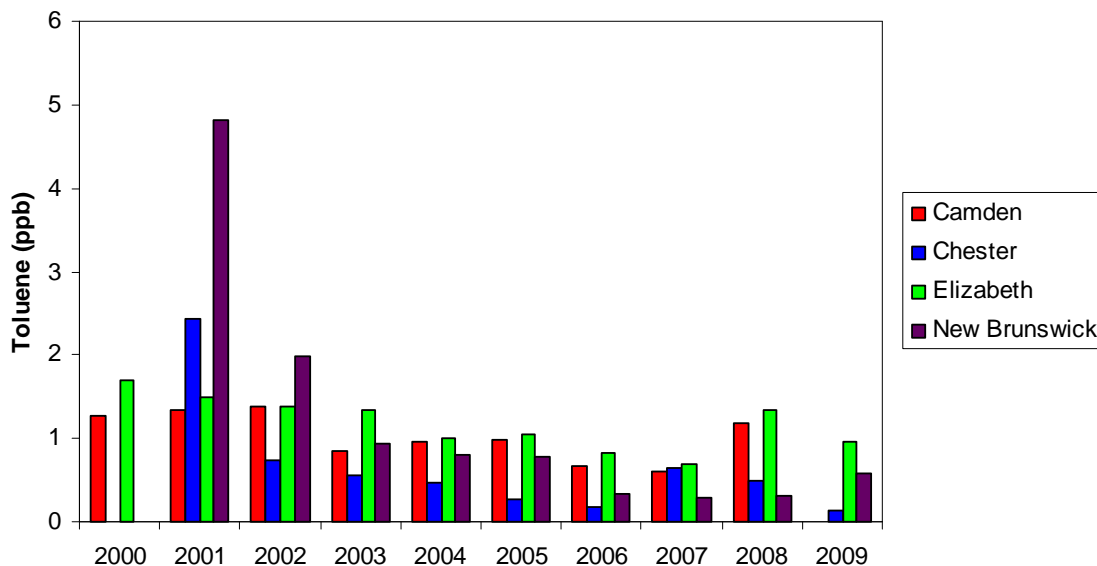
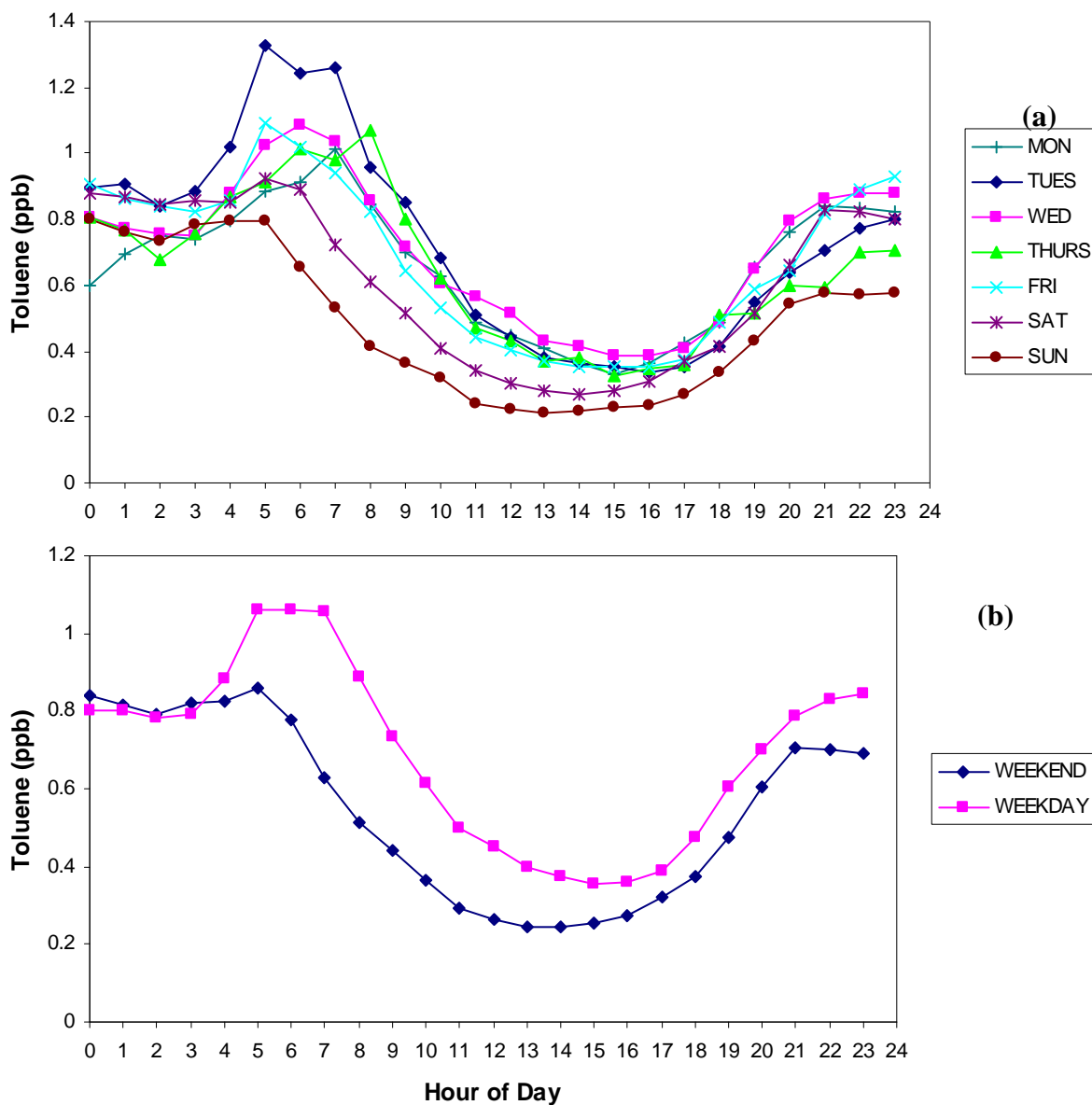
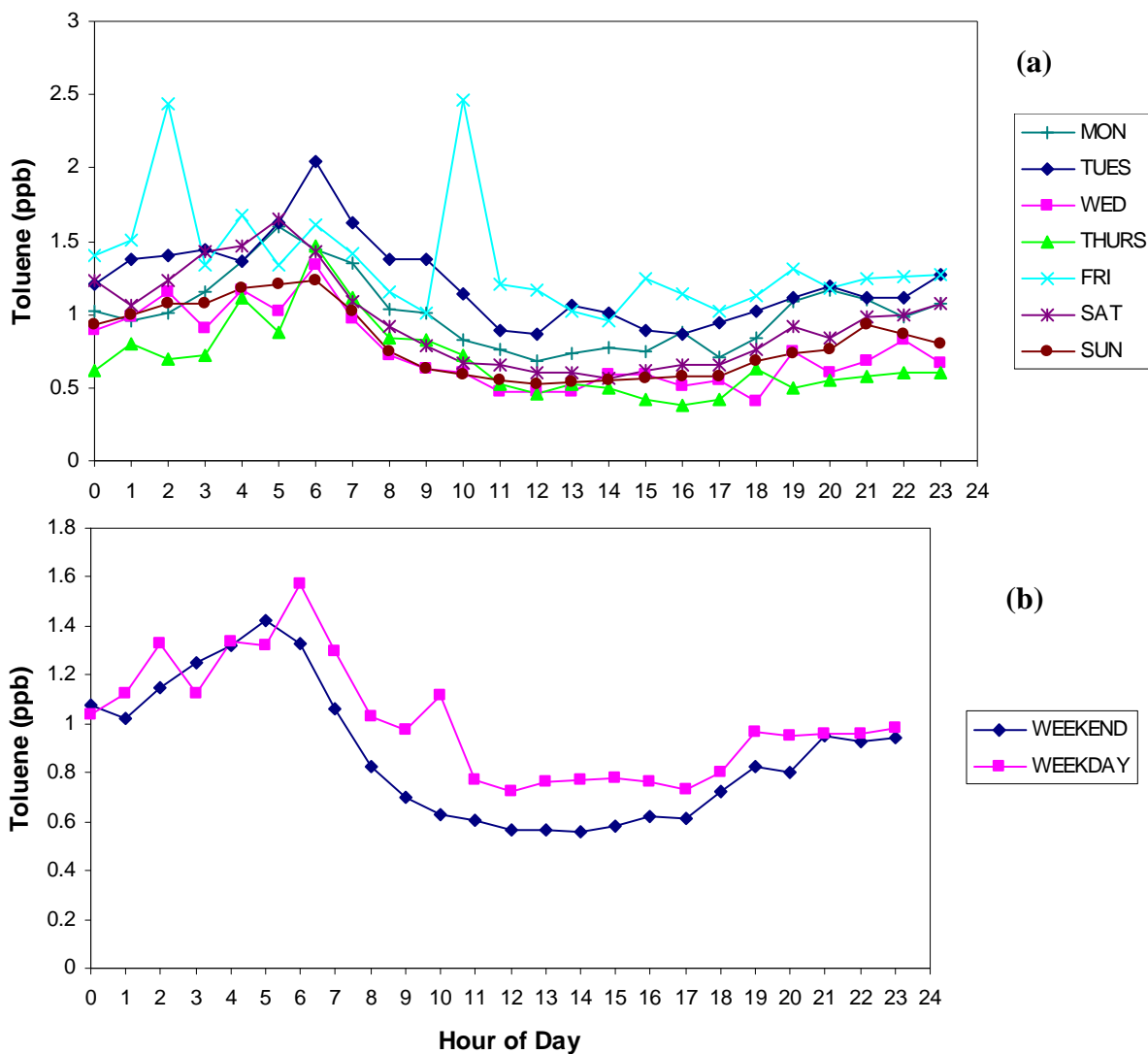


Figure 3-36. Comparison of average hourly toluene concentrations for 2000-2008 at the Camden PAMS



(a) Every day of the week. (b) Weekday and weekends.

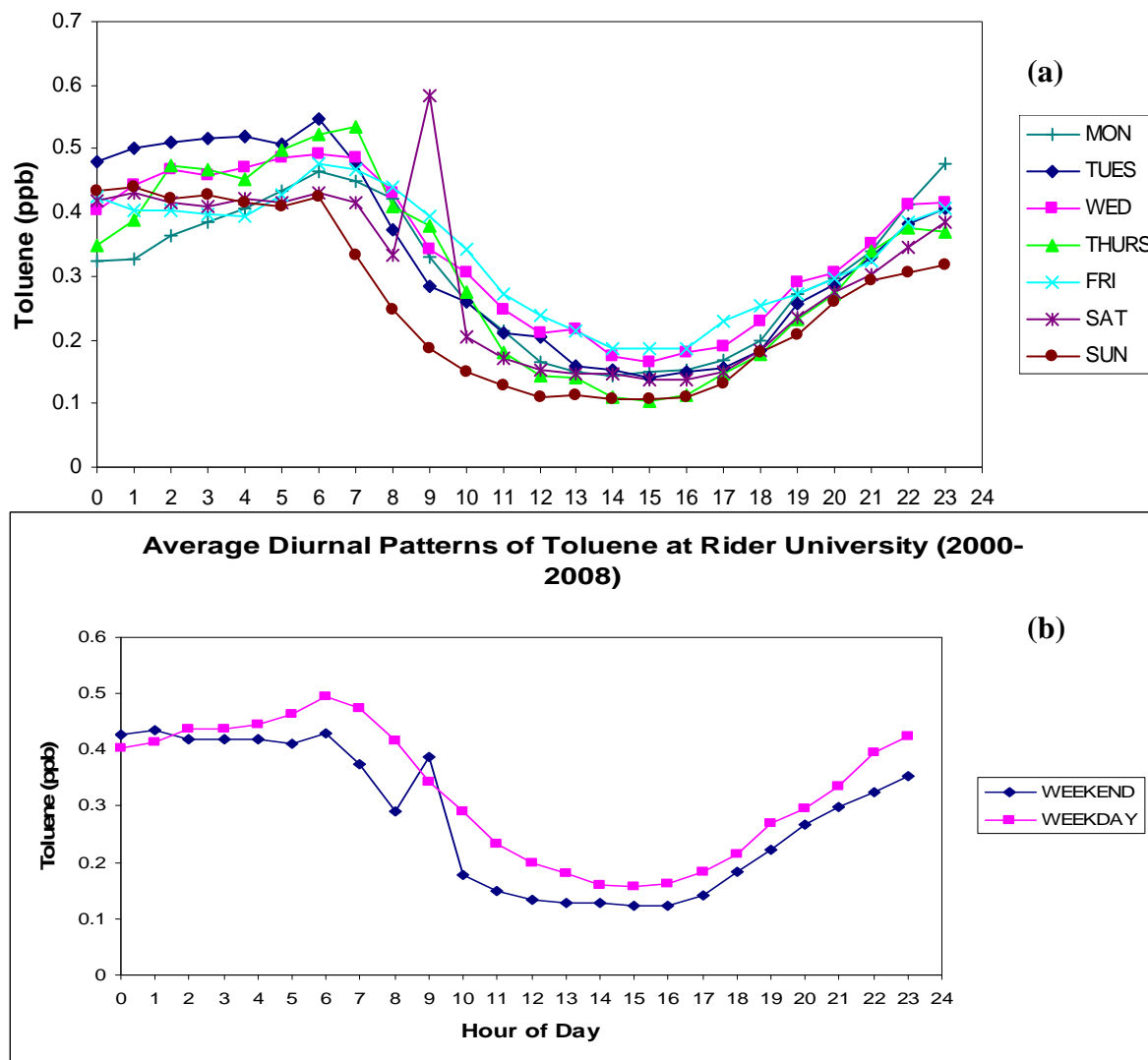
Figure 3-37. Comparison of average hourly toluene concentrations for 2000-2008 at the Rutgers University PAMS



(a) Every day of the week. (b) Weekday and weekends.

Note: The peaks for hours 2 and 10 on Friday are due to several events where monitored concentrations were much higher than typical for those times.

Figure 3-38. Comparison of average hourly toluene concentrations for 2000-2008 at the Rider University PAMS



(a) Every day of the week. (b) Weekday and weekends.

Note: The peak for hour 9 on Saturday is due in particular to one event where the monitored concentration was much higher (approximately 600 times more) than typical for that time.

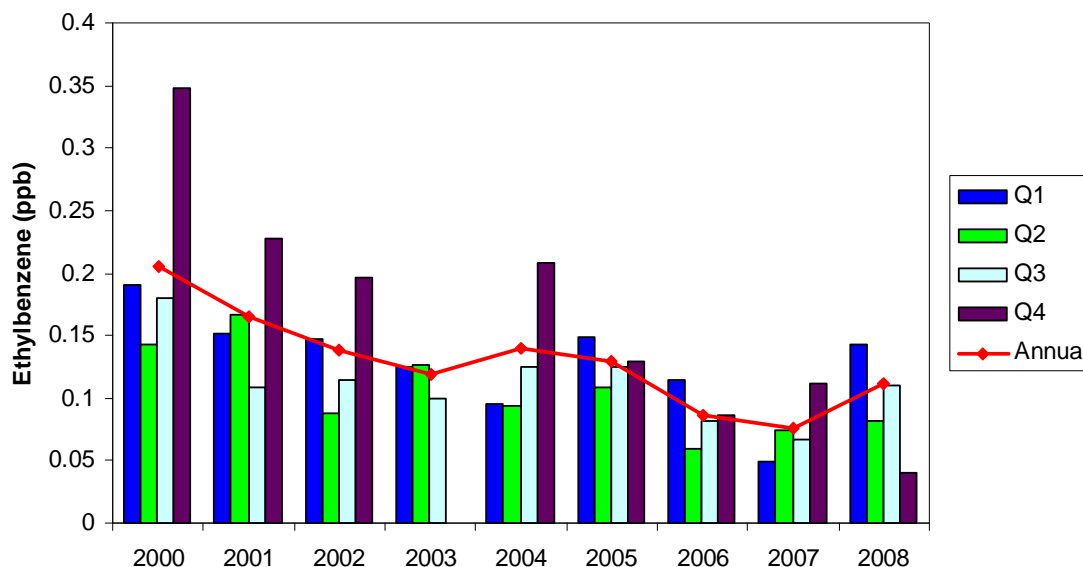
Ethylbenzene

At each of the four measurement sites, ethylbenzene followed a decreasing trend until 2008, when there was a slight upturn, as seen in Figure 3-39 through Figure 3-42. As with toluene and xylenes, the quarterly behavior was variable. While at Camden, the fourth quarter most often had the highest concentration, the third quarter most often saw the highest concentration at New Brunswick and Chester. While there were many years in Figure 3-43 when Camden winter concentrations exceeded summer concentrations, at other sites the opposite was true. There were also many years when summer and winter concentrations were similar. While 9.7 percent of the ethylbenzene measurements were below the MDL, most of those sub-MDL concentrations were measured in earlier years.

Figure 3-44 shows the comparison of annual average ethylbenzene across the four long-term New Jersey air toxics measurement sites. Camden and Elizabeth had similar ambient ethylbenzene levels for most years, yet Elizabeth typically had the highest annual average concentration. New Brunswick had the highest annual average concentration in 2001 and 2002. After 2001, Chester consistently had the lowest annual average ethylbenzene concentration of all four sites.

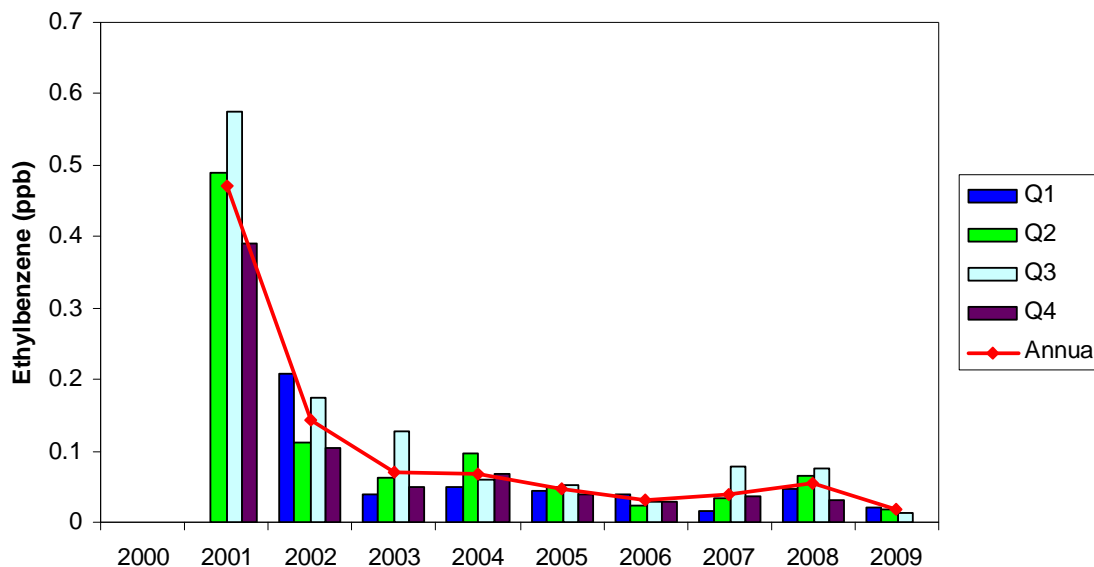
The diurnal behavior of ethylbenzene is illustrated in Figure 3-45 through Figure 3-47, which contain the hourly average ethylbenzene concentrations measured at the PAMS sites at Camden, Rutgers University, and Rider University. In the (a) plots, the average hourly concentrations for each day of the week are given. The (b) plots show the average weekday and weekend concentrations. Only days with 24 hours of measurements were used in calculating the averages. For most sites, the average peak in ethylbenzene concentrations is around 5:00 or 6:00 AM, with the exception of the average weekday peak at Rider University, which is at 7:00 AM. Weekends do not see as much of an early morning peak as weekdays, and 5:00 or 6:00 AM is either only slightly higher than or at the same level as the previous hours. Ethylbenzene levels go down at all sites over the course of the day, reaching a minimum in the early afternoon that is typical of species consumed in the atmosphere by hydroxyl radicals or direct photolysis. Ethylbenzene concentrations rise again starting in the early evening, as the sun goes down. Weekend ethylbenzene concentrations are typically lower than corresponding weekday concentrations. As with similar species, the early morning peak is likely due to morning mobile source emissions and the trough is likely due to reactions with the hydroxyl radical and a higher mixing height in the afternoon.

Figure 3-39. Quarterly and annual average ethylbenzene concentrations for 2000-2008 at Camden



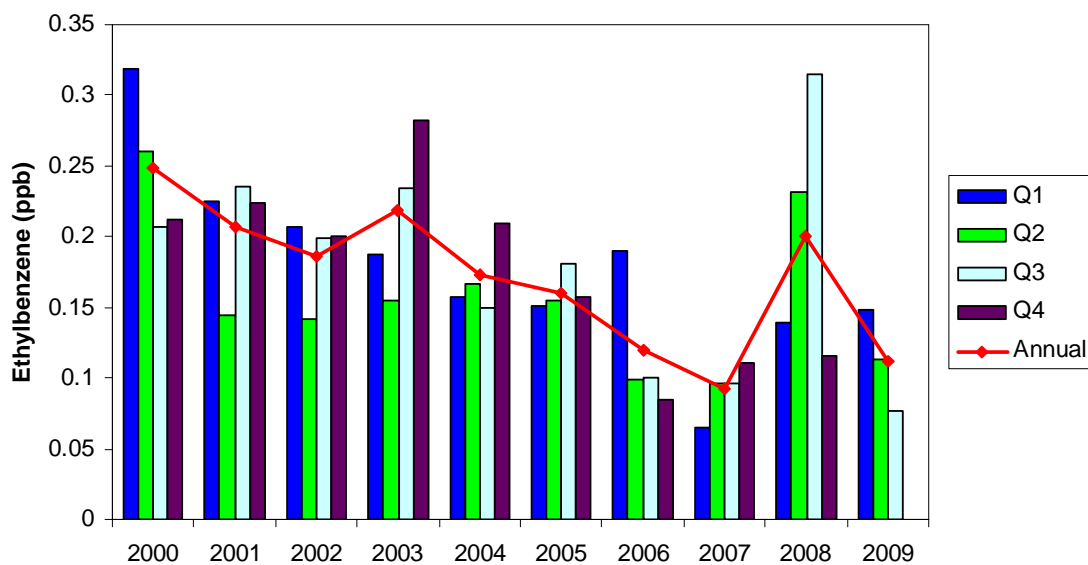
Note: Data were not available for 2009 or the last quarter of 2003 at Camden.

Figure 3-40. Quarterly and annual average ethylbenzene concentrations for 2001-2009 at Chester



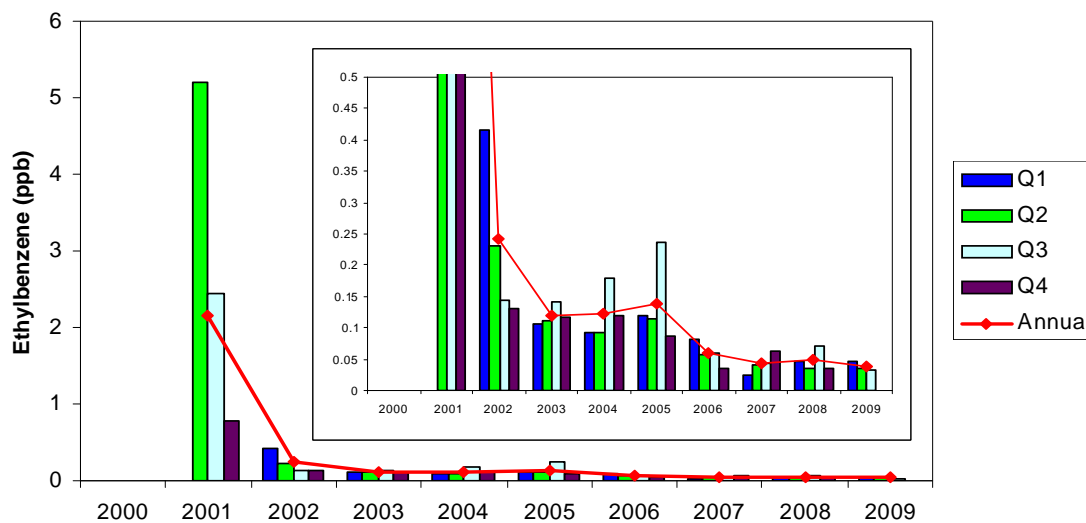
Note: At the time of retrieval, data were not available for the last quarter of 2009 at Chester. Chester came online in 2001.

Figure 3-41. Quarterly and annual average ethylbenzene concentrations for 2000-2009 at Elizabeth



Note: At the time of retrieval, data were not available for the last quarter of 2009 at Elizabeth.

Figure 3-42. Quarterly and annual average ethylbenzene concentrations for 2000-2009 at New Brunswick



Note: At the time of retrieval, data were not available for the last quarter of 2009 at New Brunswick.

Figure 3-43. Summer and winter average ethylbenzene concentrations at Camden, Chester, Elizabeth, and New Brunswick

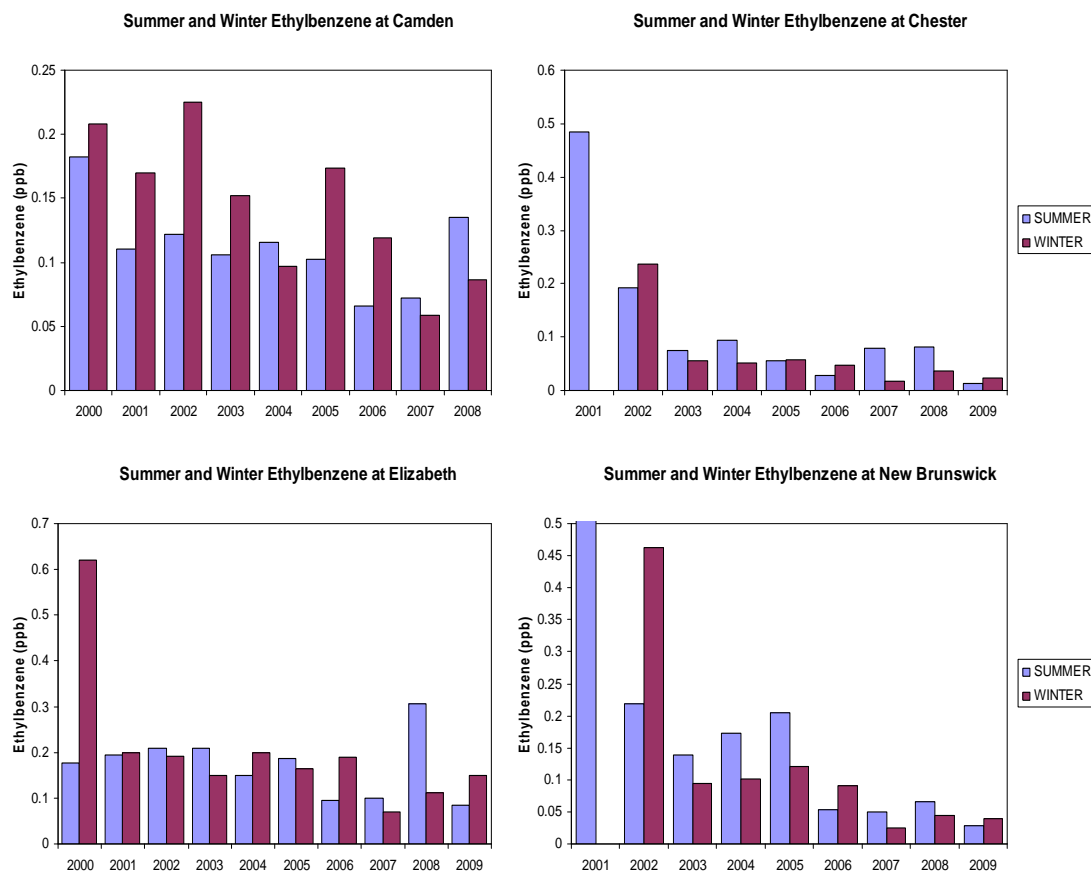


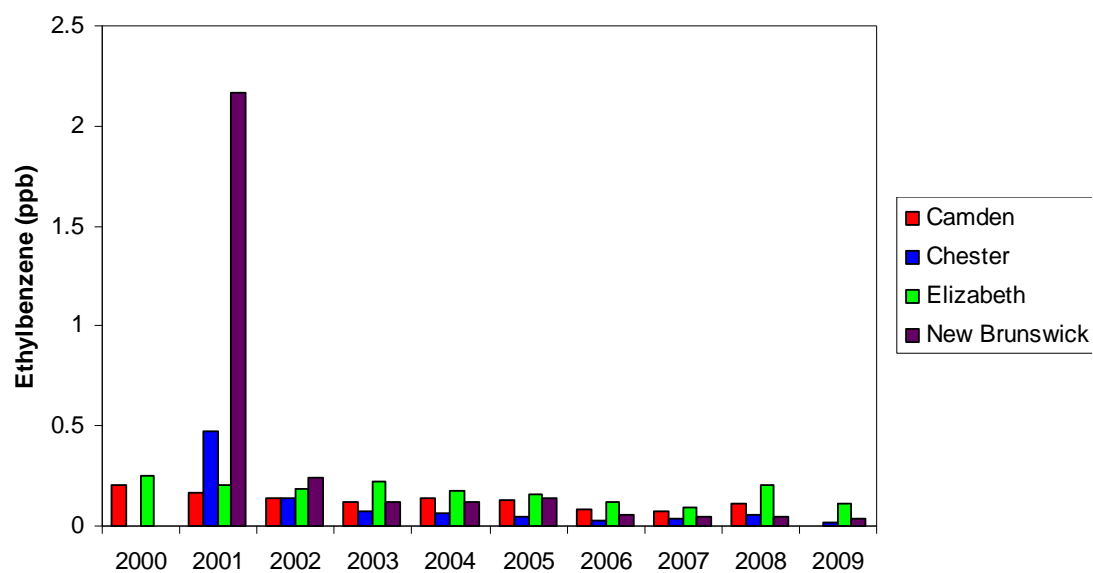
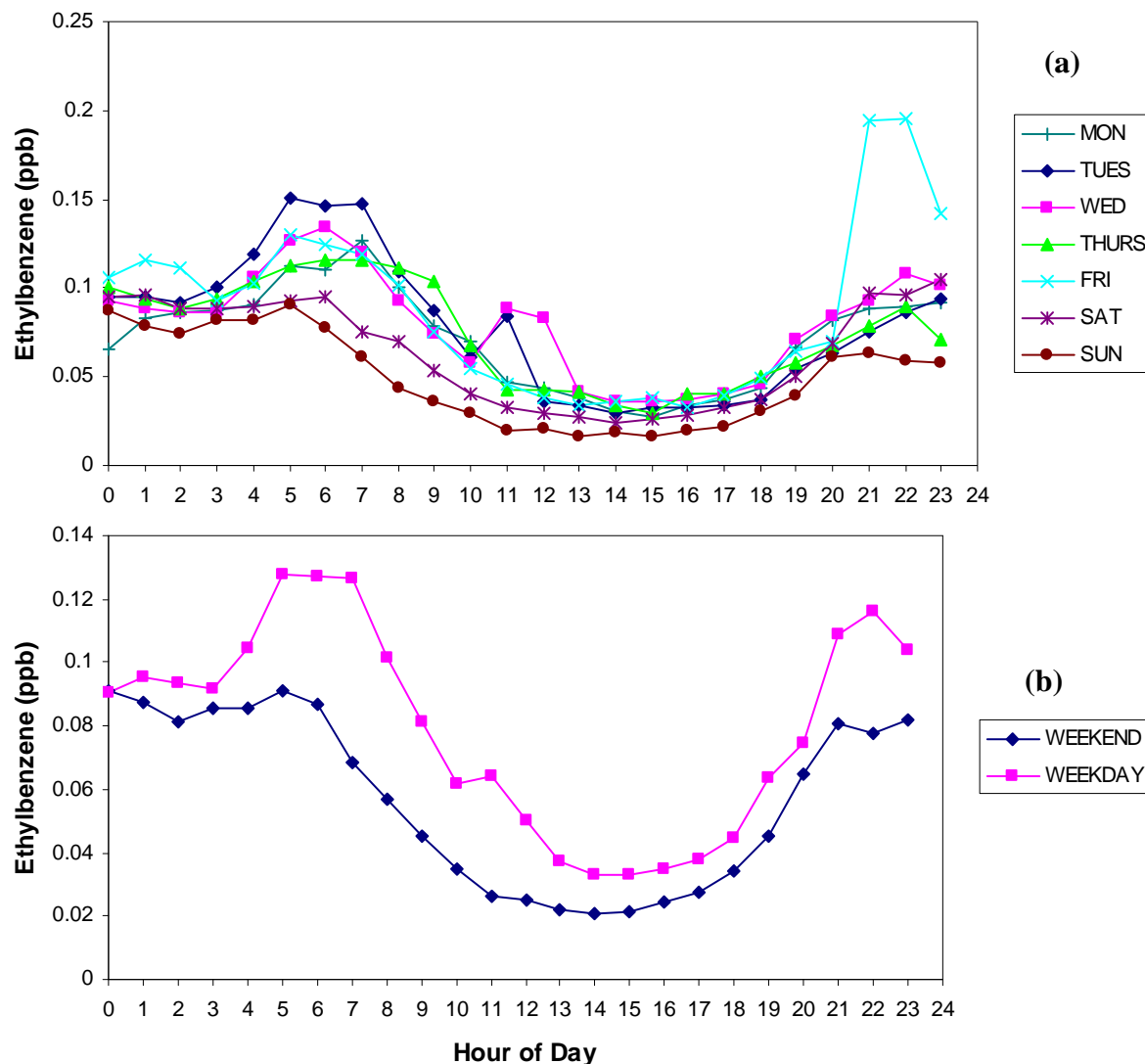
Figure 3-44. Comparison of annual average ethylbenzene concentrations by site

Figure 3-45. Comparison of average hourly ethylbenzene concentrations for 2000-2008 at the Camden PAMS



(a) Every day of the week. (b) Weekday and weekends.

Note: The peak for hours 21-23 on Friday is due to several events where monitored concentrations were much higher than typical for those times.

Figure 3-46. Comparison of average hourly ethylbenzene concentrations for 2000-2008 at the Rutgers University PAMS

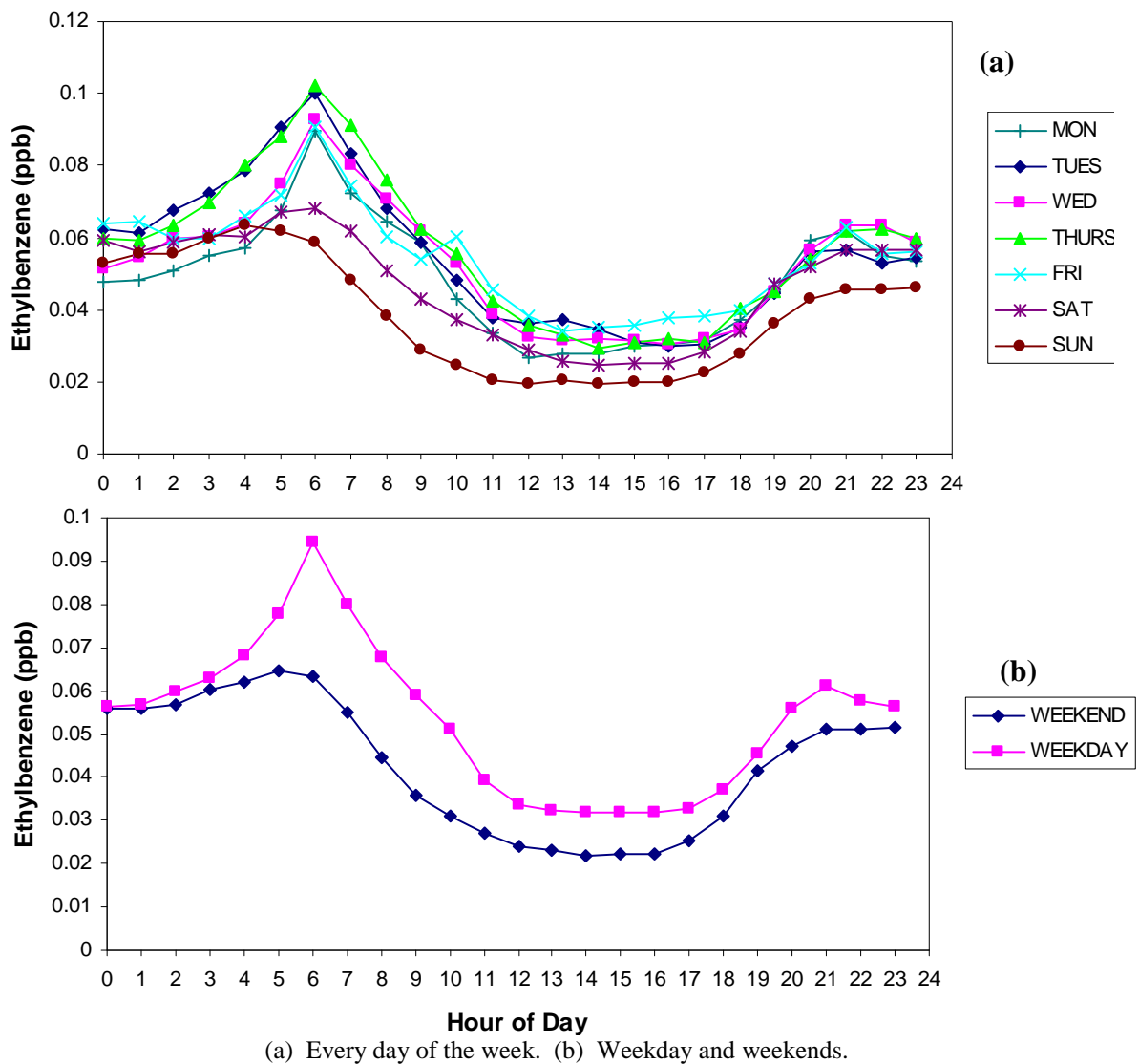
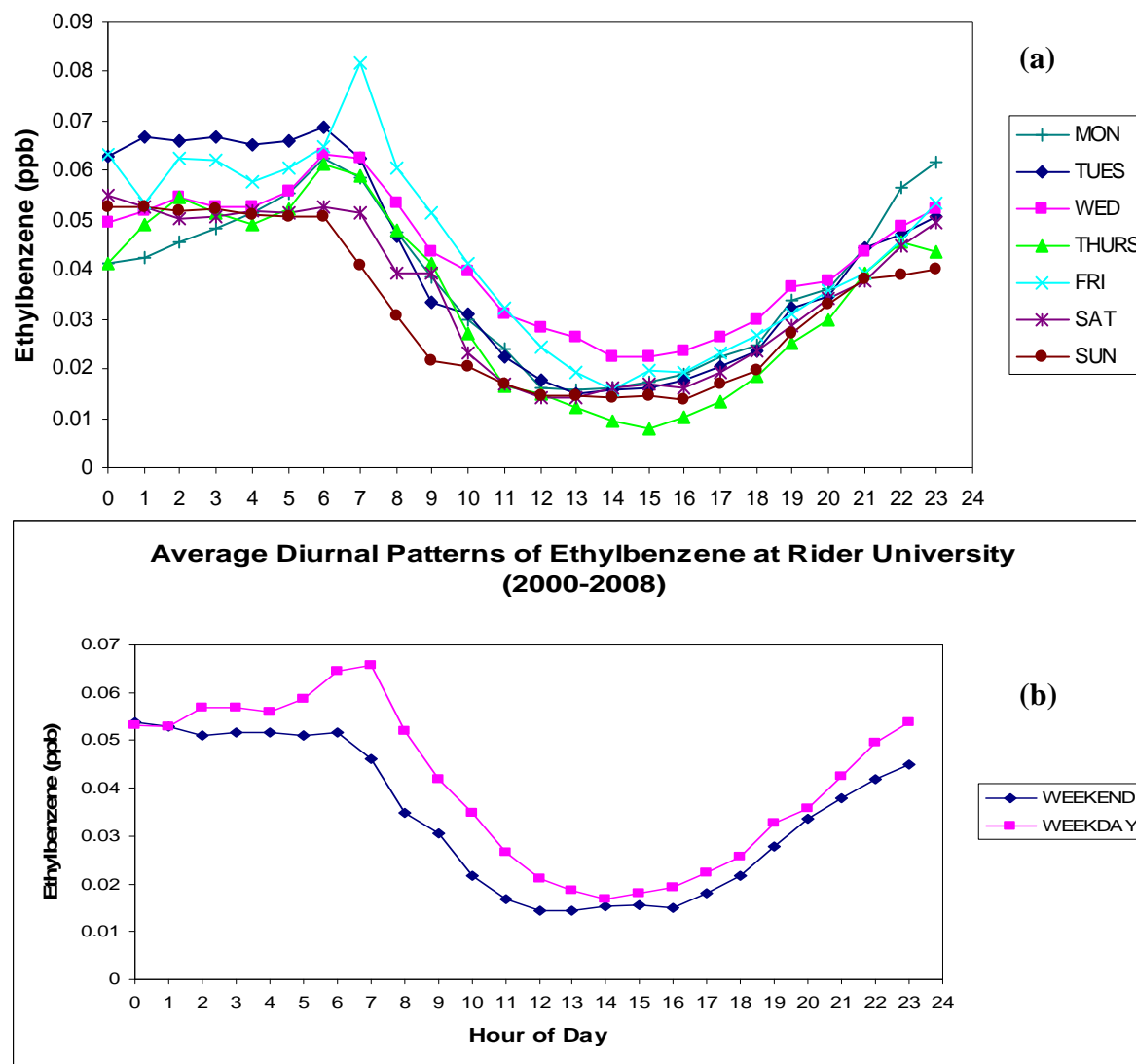


Figure 3-47. Comparison of average hourly ethylbenzene concentrations for 2000-2008 at the Rider University PAMS



(a) Every day of the week. (b) Weekday and weekends.

Group B compounds

Group B compounds are characterized as having limited emissions, are very slowly depleted in the atmosphere, are well-mixed, and have long atmospheric lifetimes. Therefore, these compounds are likely to be rather uniform in both spatial and temporal distribution. Carbon tetrachloride is the only species in Group B for the purposes of this analysis.

In 2005, there were no sources that emitted more than 1 ton of carbon tetrachloride within New Jersey. In the same year, 7 sources accounted for the majority of domestic carbon tetrachloride emissions, and only 34 sources emitted more than 1 ton per year, accounting for 89 percent of domestic emissions. Texas (26 percent) and Louisiana (21 percent) accounted for nearly half of all domestic emissions. Because there are few emission sources of carbon tetrachloride in and around New Jersey, monitors in the region are influenced by regional, national, and international sources, and therefore all

Table 3-4. Summary of domestic carbon tetrachloride emissions in 2005

State	Total emissions of carbon tetrachloride (tons per year)	Count of source facilities
Texas	56.4	324
Louisiana	45.2	248
Alabama	13.1	312
South Carolina	9.5	102
Oregon	9.4	262
Wisconsin	8.7	209
Oklahoma	8.5	119
Minnesota	8.3	717
Kansas	7.3	127
Virginia	6.9	265
Kentucky	6.7	217
MO	6.1	81
Ohio	5.1	136
Arkansas	5.1	78
Maine	4.7	99
North Carolina	3.2	331
Mississippi	3.0	120
California	2.4	3,451
Pennsylvania	2.3	37
Illinois	1.2	372
Tennessee	0.8	199
Utah	0.7	23
West Virginia	0.6	79
Colorado	0.5	782
Michigan	0.4	1,349
Iowa	0.4	85
Washington	0.3	81
Nebraska	0.2	43
Florida	0.2	234
Maryland	0.2	44
New York	0.1	731
Vermont	0.1	52
New Hampshire	0.1	42

Note: States are ordered from highest to lowest aggregate carbon tetrachloride emissions. Emissions are rounded to the nearest 0.1 tons per year. States with total emissions below 0.05 are omitted from the list.
Source: 2005 National Emissions Inventory, US EPA (2008).

sites had similar concentration levels (US EPA, 2008).

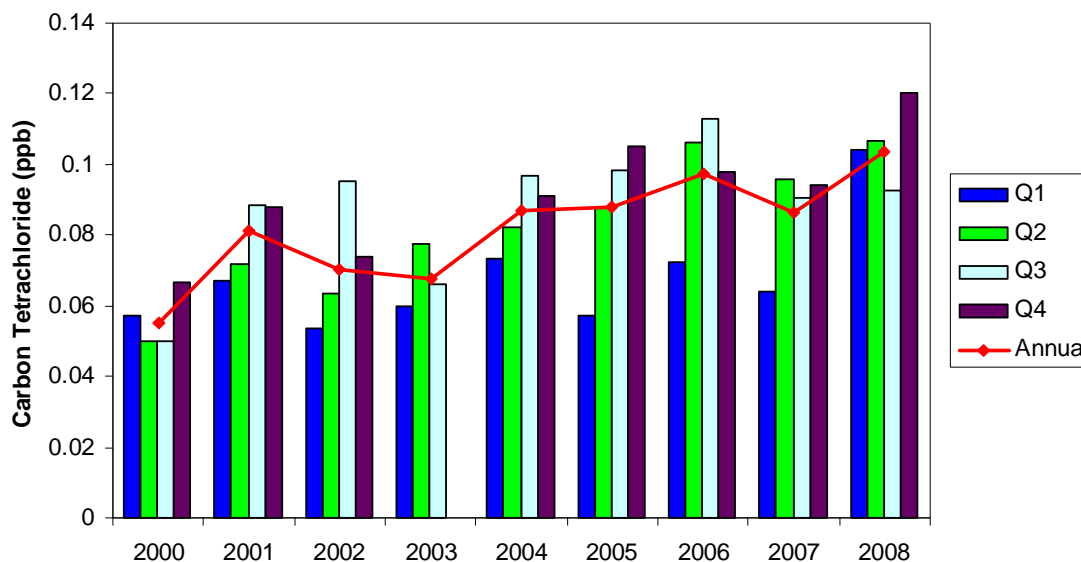
Carbon tetrachloride

Annual and quarterly average carbon tetrachloride concentrations at the four air toxic measurement sites are given in Figure 3-48 through Figure 3-51. For the years examined here, 28 percent of the carbon tetrachloride measurements were below the detection limit; however, the percentage of measurements above the MDL showed an increase since 2004. Only three values were below the MDL between 2006 and 2009. As a result, the pre-2004 concentrations are significantly influenced by the value for half of the MDL in the quarterly and annual average figures.

Though the concentrations fluctuated from year to year, there was an apparent overall upward trend for carbon tetrachloride concentrations at each site. The reason for this upward trend is not clear, as emissions sources for carbon tetrachloride in the United States are limited, and many uses of carbon tetrachloride have been discontinued over the years. The rising concentrations may be due to rising global levels of carbon tetrachloride emissions. Also, because the atmospheric lifetime of carbon tetrachloride is very long (decades), even small, distant emission sources may be able to sustain and even increase ambient levels, though the budget of carbon tetrachloride is still poorly understood (Allen et al., 2009). The relative concentration levels between the quarters varied from year to year; however, the first quarter most often had the lowest quarterly concentration. The third quarter most frequently had the highest quarterly average concentration of carbon tetrachloride (Figure 3-52). At each site, summer concentrations of carbon tetrachloride were typically higher than winter concentrations for most years.

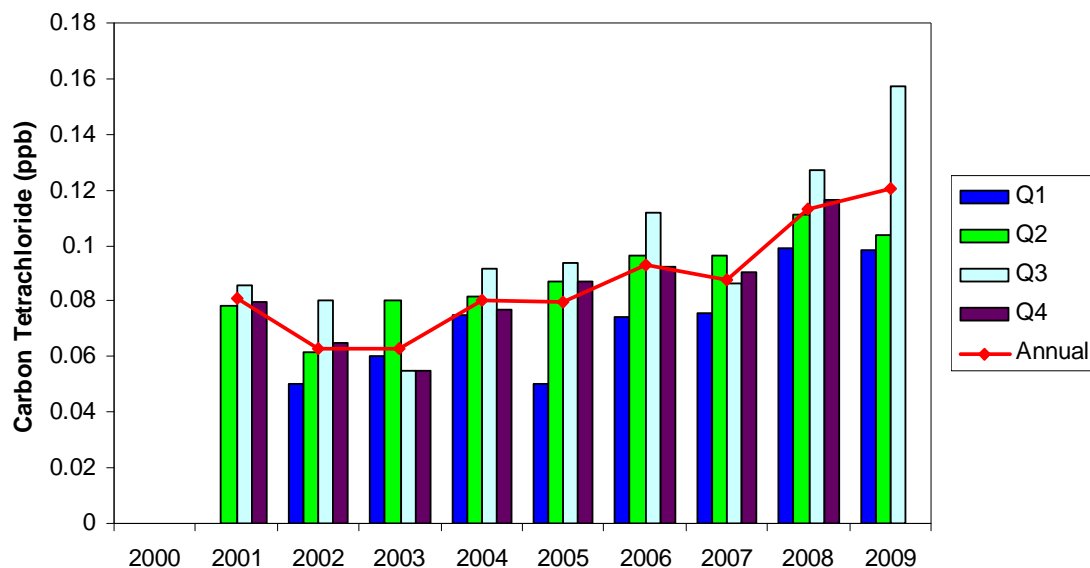
Figure 3-53 compares the level of annual average carbon tetrachloride at each measurement site. As might be expected for a long-lived, well-mixed chemical, the concentrations were similar for all sites in a given year.

Figure 3-48. Quarterly and annual average carbon tetrachloride concentrations for 2000-2008 at Camden



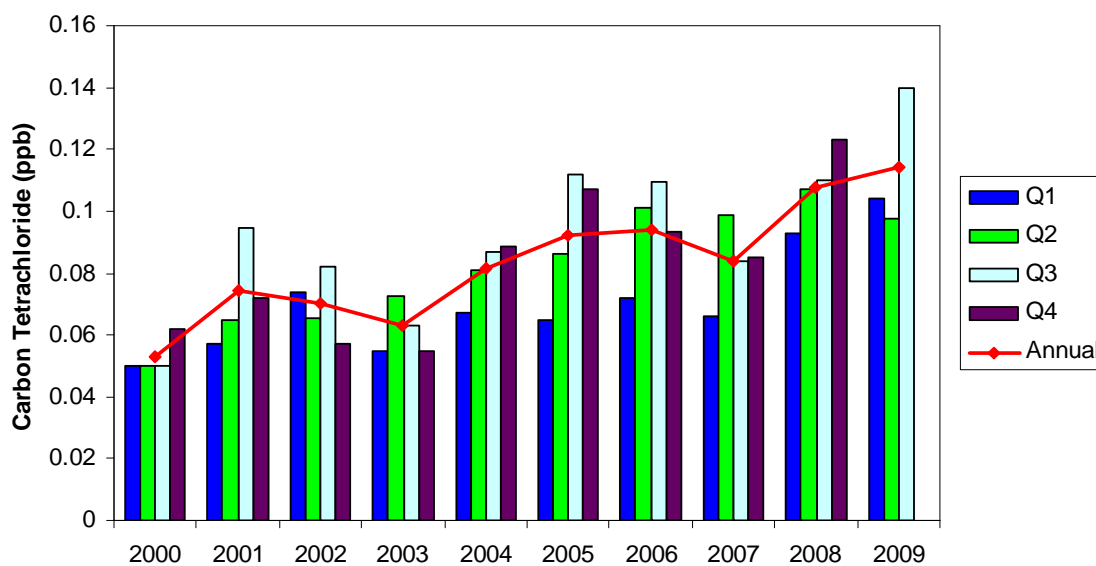
Note: Data were not available for 2009 or the last quarter of 2003 at Camden.

Figure 3-49. Quarterly and annual average carbon tetrachloride concentrations for 2001-2009 at Chester



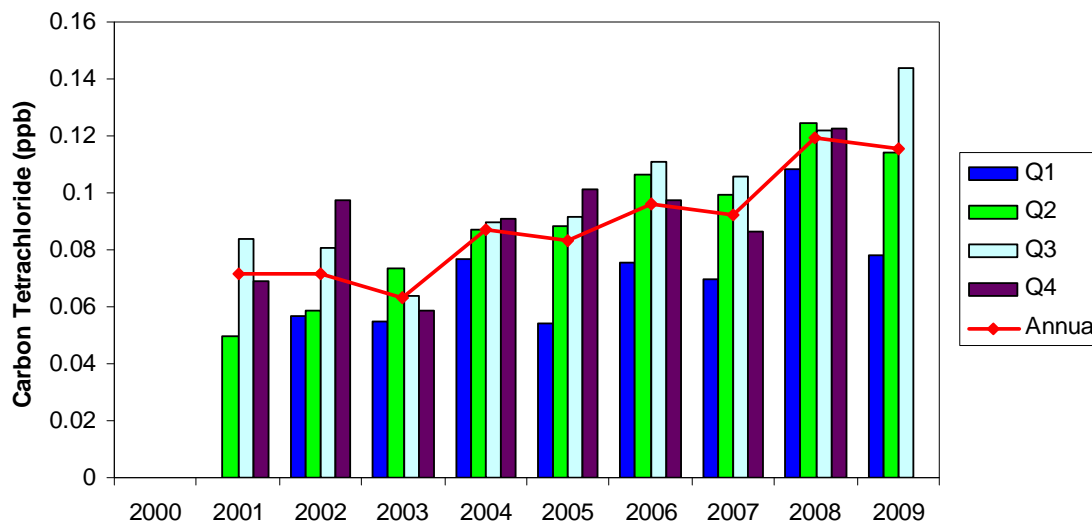
Note: At the time of retrieval, data were not available for the last quarter of 2009 at Chester. Chester came online in 2001.

Figure 3-50. Quarterly and annual average carbon tetrachloride concentrations for 2000-2009 at Elizabeth



Note: At the time of retrieval, data were not available for the last quarter of 2009 at Elizabeth.

Figure 3-51. Quarterly and annual average carbon tetrachloride concentrations for 2000-2009 at New Brunswick



Note: At the time of retrieval, data were not available for the last quarter of 2009 at New Brunswick.

Figure 3-52. Summer and winter average carbon tetrachloride concentrations at Camden, Chester, Elizabeth, and New Brunswick

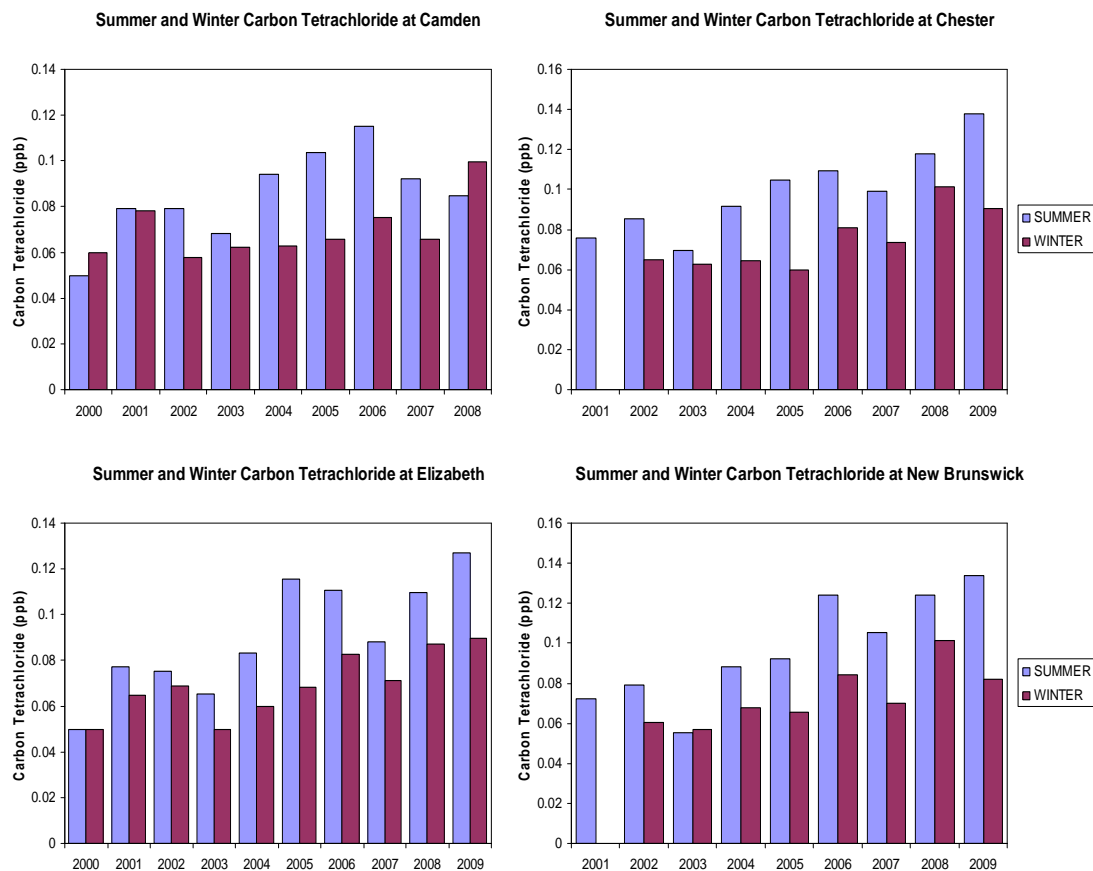
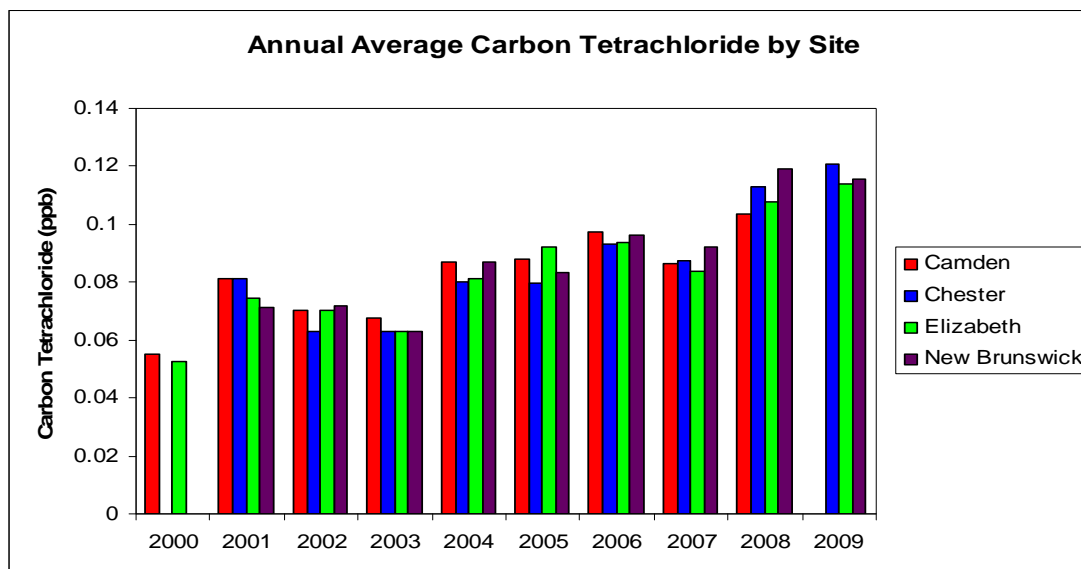


Figure 3-53. Comparison of annual average carbon tetrachloride concentrations by site



Group C compounds

Group C compounds are directly emitted into the atmosphere, readily removed by direct and/or indirect photolysis in the atmosphere, and also produced in the atmosphere through chemical reactions.

Formaldehyde

The long-term temporal “trends” in formaldehyde vary between sites (Figure 3-54 through Figure 3-57). Similar to acetaldehyde, the annual average formaldehyde concentration in Camden dropped from 2000 to 2003, spiked in 2004 and was level for 2005 through 2008, with only slight fluctuation. At the Chester site, there was a bumpy downward trend between 2000 and 2006. Concentrations rose between 2006 and 2007, and the 2008 formaldehyde concentration was only slightly lower. At the Elizabeth site, formaldehyde dropped between 2000 and 2002, then jumped significantly in 2003. Concentrations were level from 2004 through 2007, and in 2008, the concentration dropped again. At New Brunswick, there was a similar lack of long-term trend. The annual average concentration dropped between 2001 and 2002, was high in 2004 and 2005, and then dropped again in 2006. Only 0.11 percent of the total formaldehyde measurements for all sites and years fall below the MDL given in the AQS data file, while NJDEP has indicated that formaldehyde monitoring data from the NJ Air Toxics sites are of unknown quality (Pietarinen, 2011).

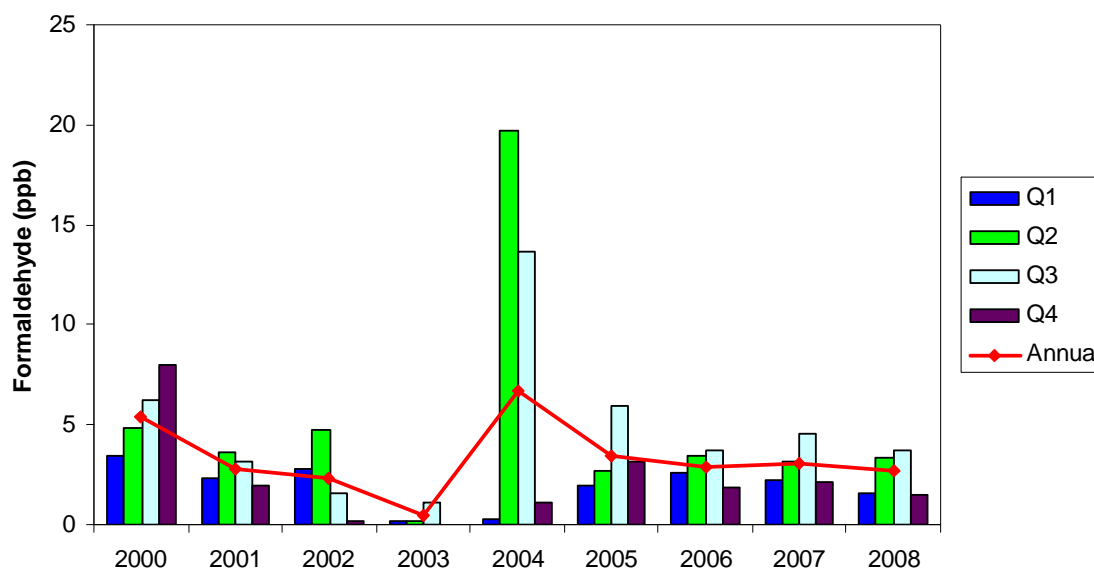
Quarterly concentrations in Figure 3-54 through Figure 3-57 indicate a clear tendency at all sites for the second and third quarters to have higher formaldehyde concentrations than the first and fourth quarters in any given year. Second and third quarter concentrations of formaldehyde at Camden are unusually high, indicating

equipment malfunction or other monitoring errors, though levels are also elevated at the Chester and New Brunswick monitors. The seasonal dependence is also evident in Figure 3-58 where summer concentrations of formaldehyde exceed winter concentrations in a given year for nearly all years and sites. While formaldehyde concentrations are influenced by direct emissions, photochemical production of formaldehyde may account for a significant fraction of ambient levels (Luecken et al., 2006). However, it is possible that this seasonal trend may also be an artifact of the data quality issue discussed in Section 3.3. A higher formaldehyde concentration is likely observed in the warmer months due to the higher temperatures, photolysis rates, and biogenic emissions.

The relative level of formaldehyde measured at each site varied from year to year (Figure 3-59). Each site had the maximum annual average formaldehyde concentration at least once. Of all the sites, Chester most often saw the lowest formaldehyde concentrations.

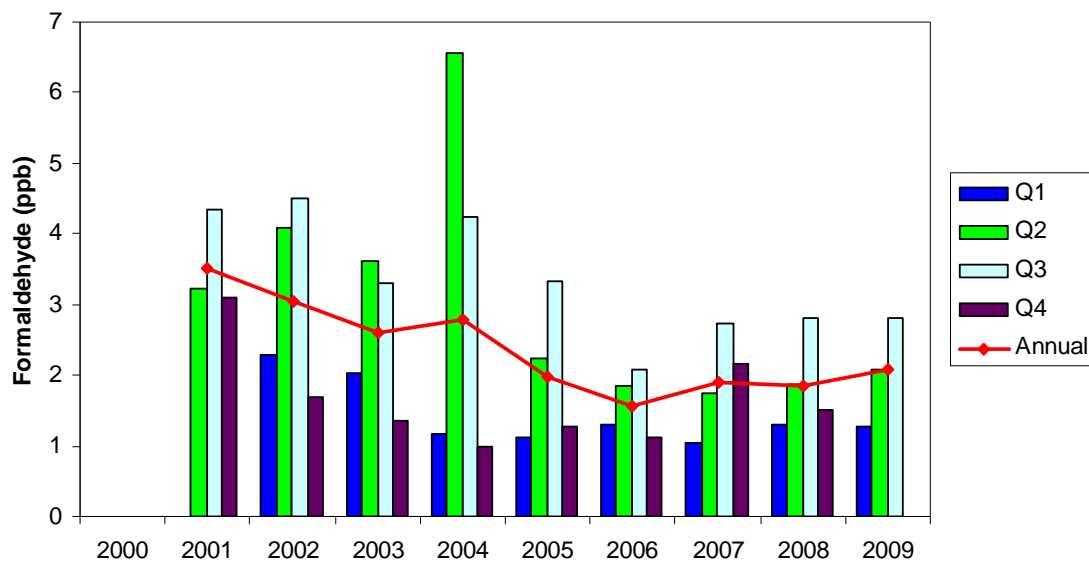
On certain days in years 2000, 2001, 2003, and 2005, 3-hour average formaldehyde concentrations were measured at the Camden PAMS. Figure 3-60 contains the weekday and weekend average formaldehyde concentrations measured at Camden. Only days for which there was a complete set of measurements were included in the average. Only weekday and weekend concentrations, rather than averages for individual days, are included here due to there being relatively few measurements. Formaldehyde concentrations show a maximum in the early afternoon hours. The weekday peak is more pronounced than the weekend peak, which is likely due to higher primary anthropogenic emissions on the weekdays. The early afternoon peak can be attributed to the combined effects of emissions and the photochemical production of formaldehyde.

Figure 3-54. Quarterly and annual average formaldehyde concentrations for 2000-2008 at Camden



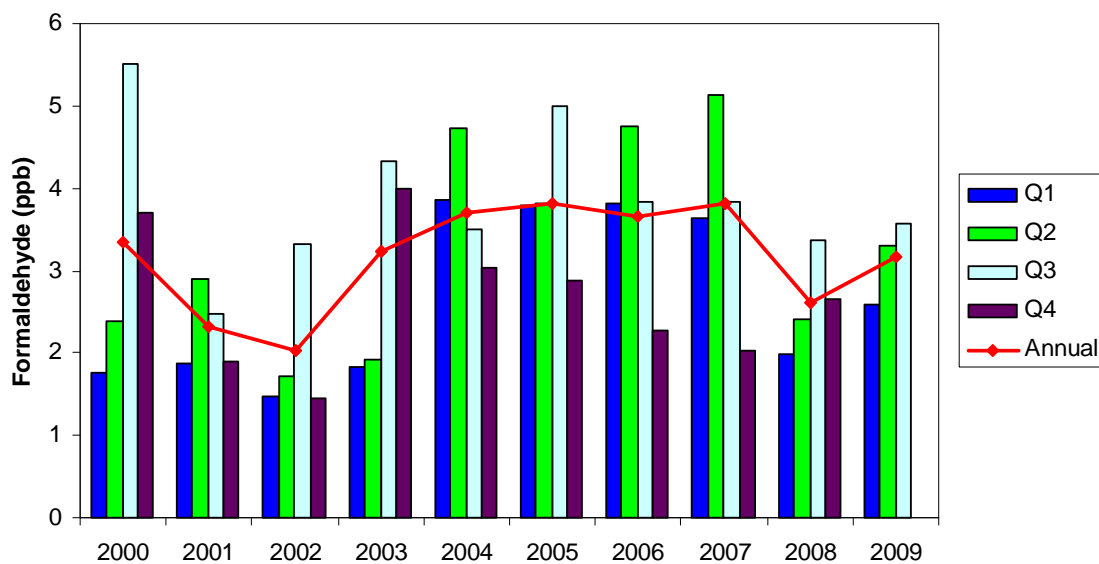
Note: Data were not available for 2009 or the last quarter of 2003 at Camden.

Figure 3-55. Quarterly and annual average formaldehyde concentrations for 2001-2009 at Chester



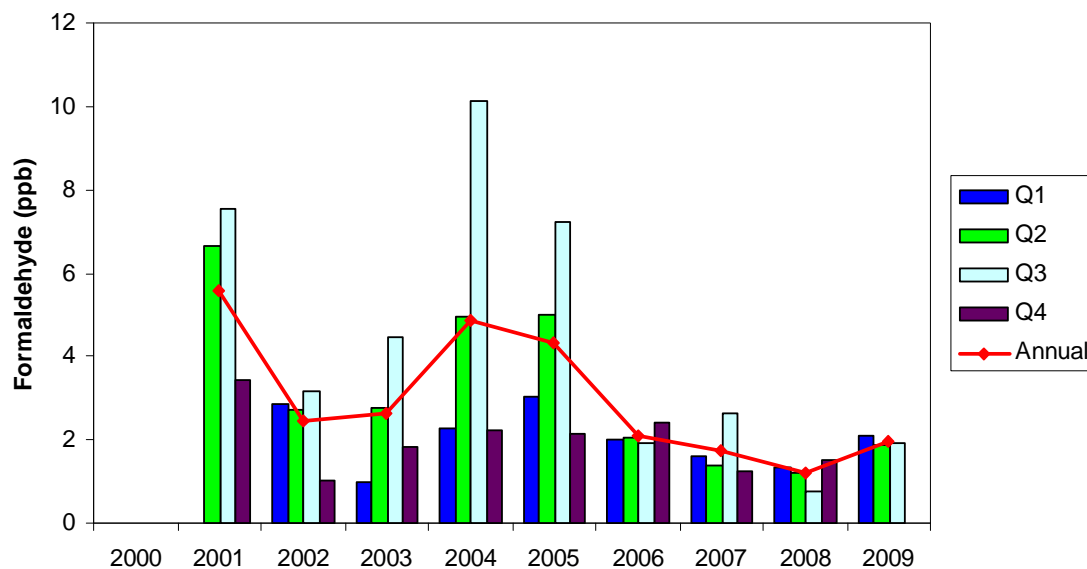
Note: At the time of retrieval, data were not available for the last quarter of 2009 at Chester. Chester came online in 2001.

Figure 3-56. Quarterly and annual average formaldehyde concentrations for 2000-2009 at Elizabeth



Note: At the time of retrieval, data were not available for the last quarter of 2009 at Elizabeth.

Figure 3-57. Quarterly and annual average formaldehyde concentrations for 2000-2009 at New Brunswick



Note: At the time of retrieval, data were not available for the last quarter of 2009 at New Brunswick.

Figure 3-58. Summer and winter average formaldehyde concentrations at Camden, Chester, Elizabeth, and New Brunswick

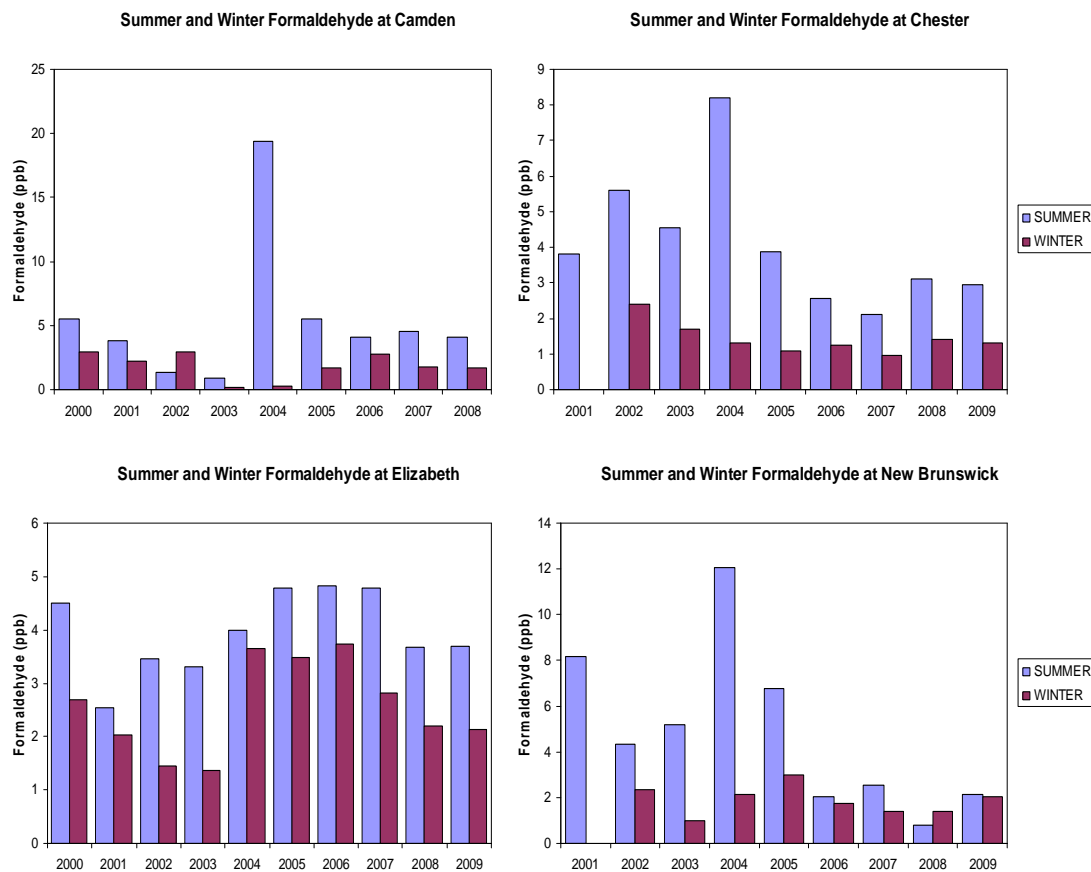


Figure 3-59. Comparison of annual average formaldehyde concentrations by site

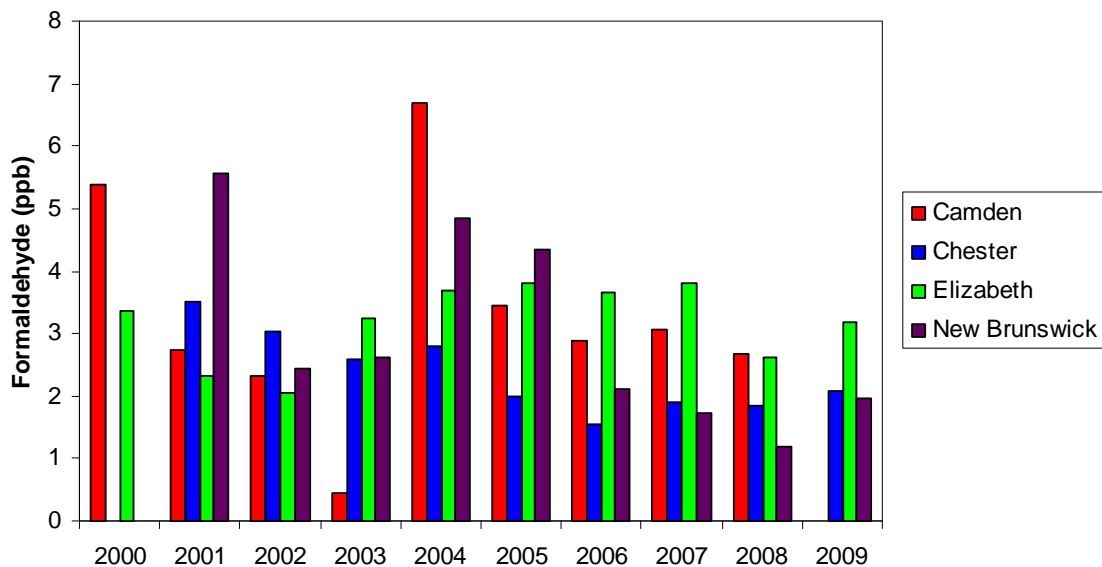
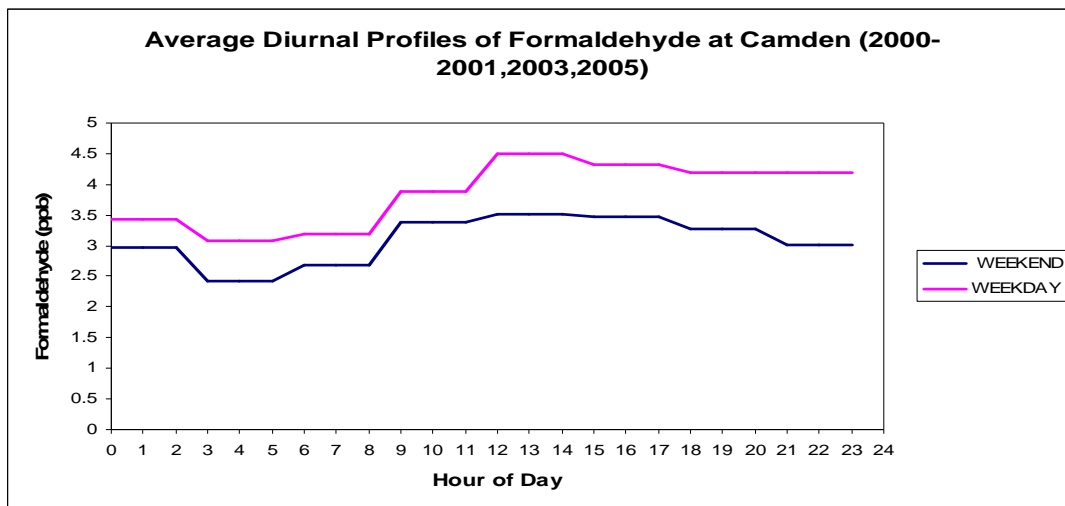


Figure 3-60. Comparison of weekday and weekend average 3-hour formaldehyde concentrations for 2000, 2001, 2003, and 2005 at the Camden PAMS



Acetaldehyde

Quarterly and annual average acetaldehyde concentrations at the four New Jersey Air Toxics sites are given in Figure 3-61 through Figure 3-64. Only 0.8 percent of the total acetaldehyde measurements for all sites and years fall below the MDL in the AQS data file, and most of those are in 2002. NJDEP has indicated that acetaldehyde monitoring data from the NJ Air Toxics sites are of unknown quality (Pietarinen, 2011).

Annual acetaldehyde concentrations did not follow a clear trend. In Camden, concentrations dropped from 2000 to 2003, spiked in 2004 (due to a high second quarter concentration that is an average of only six values), and were level for 2006 through 2008, with only slight fluctuation. At the Chester site, there was a bumpy downward trend between 2000 and 2006, and concentrations rose between 2006 and 2008. At the Elizabeth site, concentrations dropped between 2000 and 2002, jumped significantly in 2003, and then rose through 2007. In 2008, the concentration dropped down to pre-2003 levels. The annual concentration fluctuated but is similar for many years at New Brunswick with the exception of highly elevated annual concentrations in 2004 and 2005.

The quarterly behavior also varied from year to year and site to site. At New Brunswick and Elizabeth, the third quarter had the highest average concentration for most years. At Camden and Chester, it was the second quarter that most often had the highest quarterly concentration. The lowest concentrations were most often in the first or fourth quarter, but in some cases, those quarters also contained the highest. Figure 3-65 illustrates more clearly the seasonal dependence of ambient acetaldehyde concentrations at the four sites. Figure 3-65 gives the summer average and winter average⁵ acetaldehyde concentrations for each site and year. There is a clear tendency for higher concentrations

⁵ Note that year 2000 values only include January and February data, and do not include data from December of the previous year.

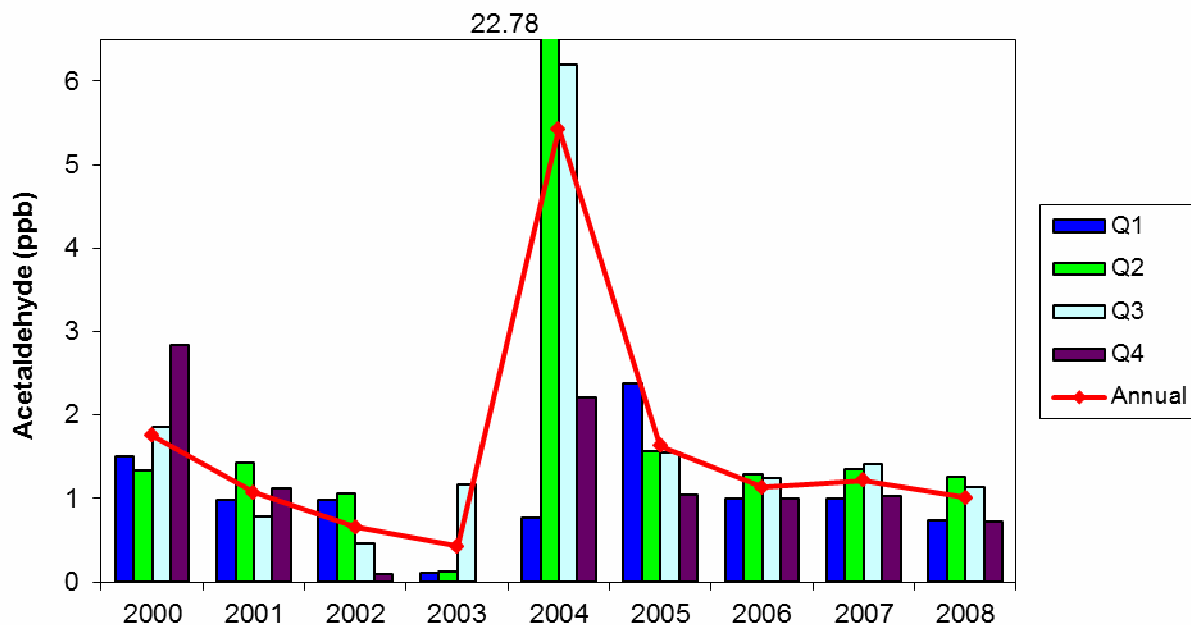
in the summer compared to winter for the same year. This holds true for most years at most sites, although there are some anomalies.

Acetaldehyde is not only depleted in the atmosphere but also chemically generated there. One might expect a higher concentration of acetaldehyde in the warmer months because biogenic emissions and higher temperatures can lead to higher acetaldehyde concentrations. That there is not always a clear quarterly trend or the fact that colder quarters sometimes see the highest concentration in a year might be due to the competing effects of atmospheric chemistry and emissions. The contributions of emissions and chemistry to ambient acetaldehyde concentrations may be of similar magnitudes, and higher direct emissions during cooler months may compensate for the lower chemical production of acetaldehyde in the atmosphere.

The annual average acetaldehyde levels varied considerably between the four sites (Figure 3-66). Each site had the maximum concentration at least once. Chester most frequently had the lowest concentration of the four sites.

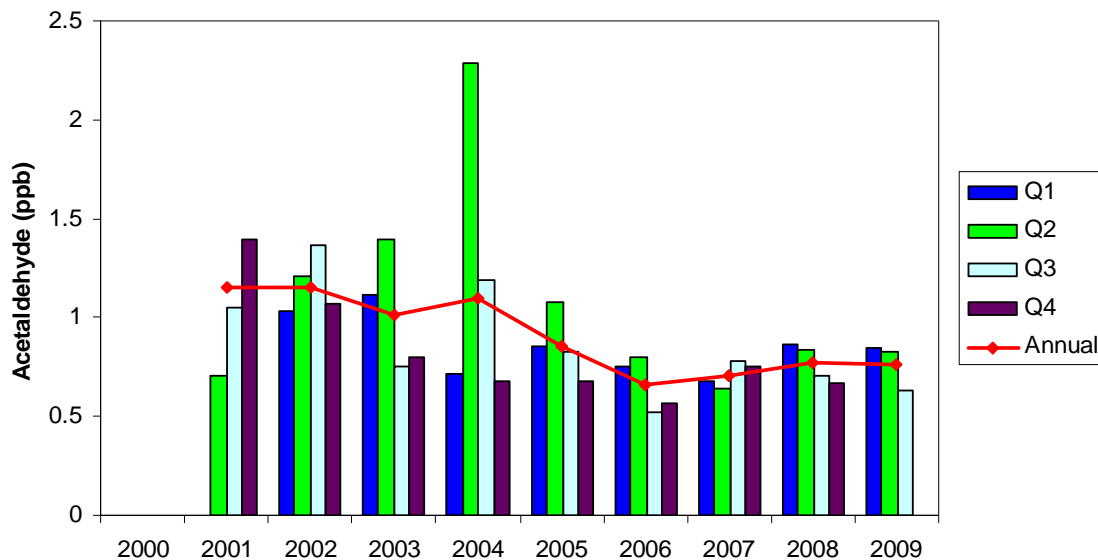
On certain days in years 2000, 2001, 2003, and 2005, 3-hour average acetaldehyde concentrations were measured at the Camden PAMS site. Figure 3-67 contains the weekday and weekend average acetaldehyde concentrations measured at Camden. Only days for which there was a complete set of measurements were included in the average. Only weekday and weekend concentrations, rather than averages for individual days, are included here due to there being relatively few measurements. Acetaldehyde concentrations show a maximum in the late morning and early afternoon hours. The weekday peak is more pronounced (and occurs later) than the weekend peak; however there is not a very large fluctuation in concentration over the course of the day for weekends or weekdays. One might expect an early afternoon peak when photochemical production is high.

Figure 3-61. Quarterly and annual average acetaldehyde concentrations for 2000-2008 at Camden



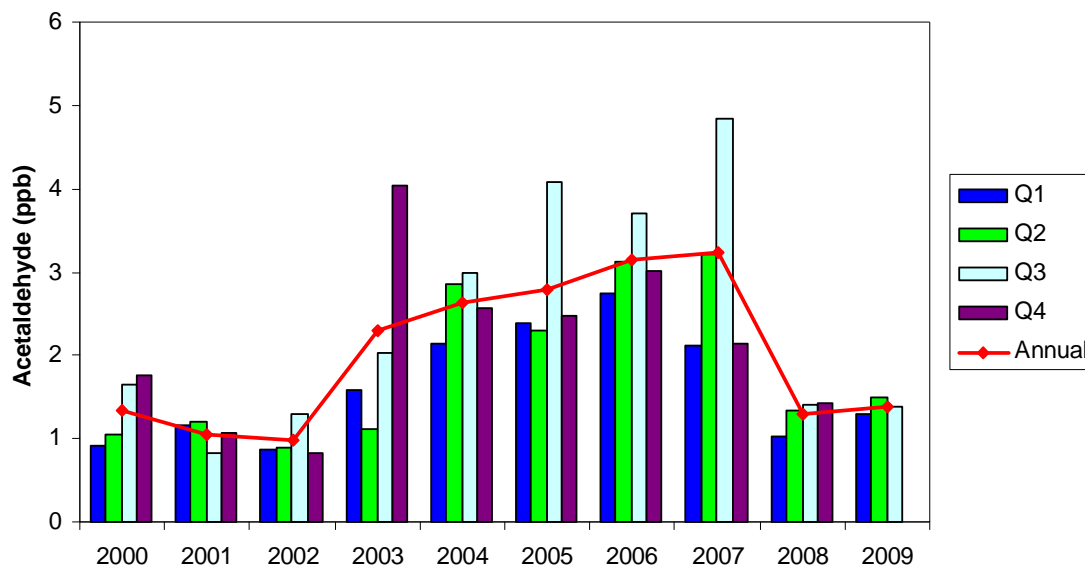
Note: Data were not available for 2009 or the last quarter of 2003 at Camden.

Figure 3-62. Quarterly and annual average acetaldehyde concentrations for 2001-2009 at Chester



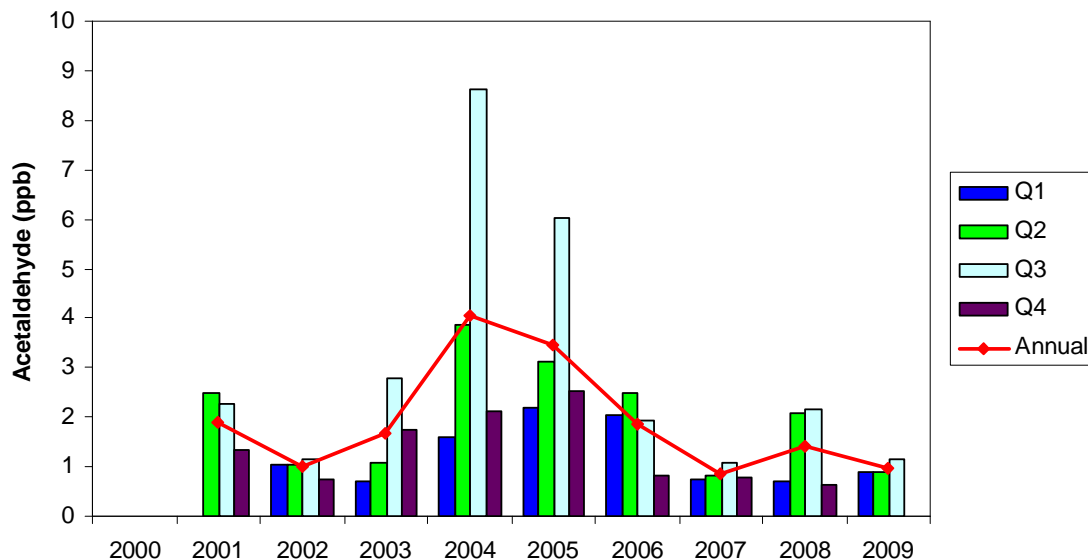
Note: At the time of retrieval, data were not available for the last quarter of 2009 at Chester. Chester came online in 2001.

Figure 3-63. Quarterly and annual average acetaldehyde concentrations for 2000-2009 at Elizabeth



Note: At the time of retrieval, data were not available for the last quarter of 2009 at Elizabeth.

Figure 3-64. Quarterly and annual average acetaldehyde concentrations for 2000-2009 at New Brunswick



Note: At the time of retrieval, data were not available for the last quarter of 2009 at New Brunswick.

Figure 3-65. Summer and winter average acetaldehyde concentrations at Camden, Chester, Elizabeth, and New Brunswick

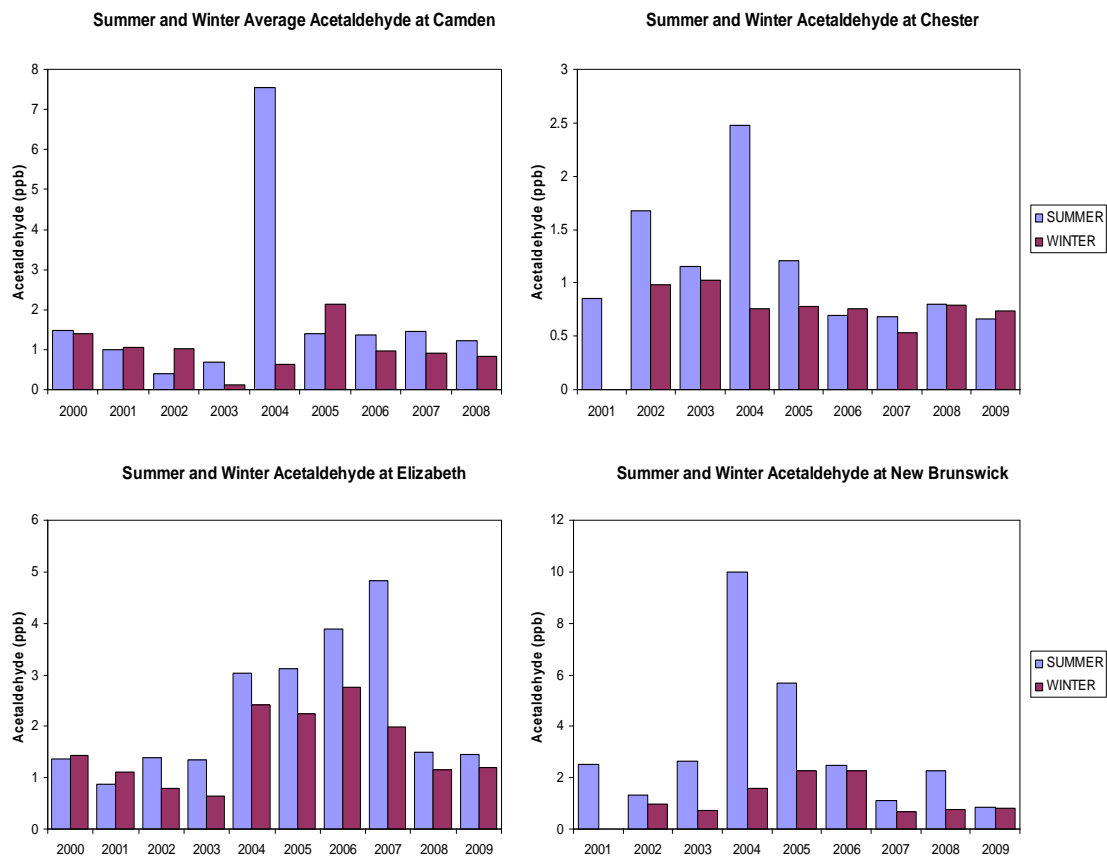


Figure 3-66. Comparison of annual average acetaldehyde concentrations by site

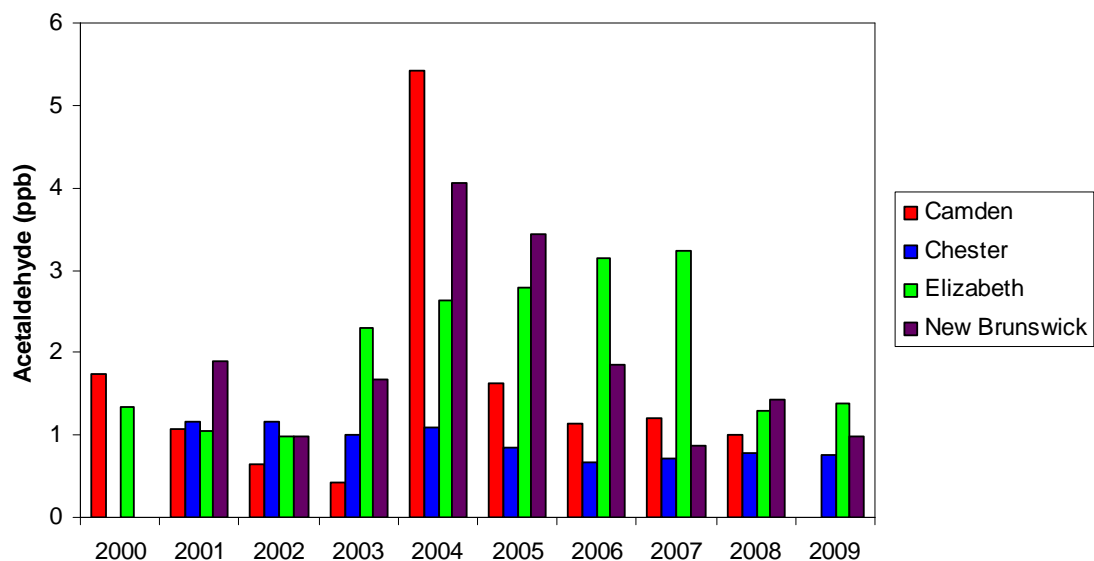
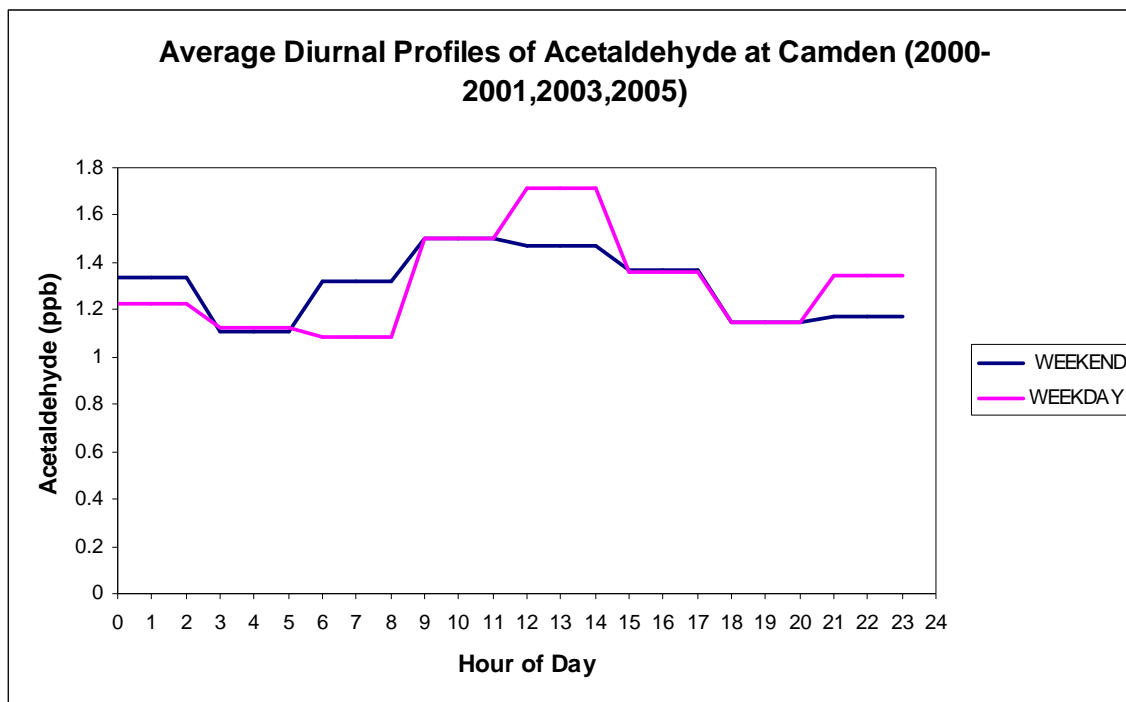


Figure 3-67. Comparison of weekday and weekend average 3-hour acetaldehyde concentrations for 2000, 2001, 2003, and 2005 at the Camden PAMS



Acrolein

There are limited data available for acrolein. Data were not available until the third quarter of 2005 (Figure 3-68 through Figure 3-71). NJDEP staff has advised against using acrolein monitoring data from the NJ Air Toxics sites (Pietarinen, 2011). Therefore, these monitoring data are discussed in generally qualitative terms and should not be interpreted as conclusive of trends or levels of acrolein in New Jersey.

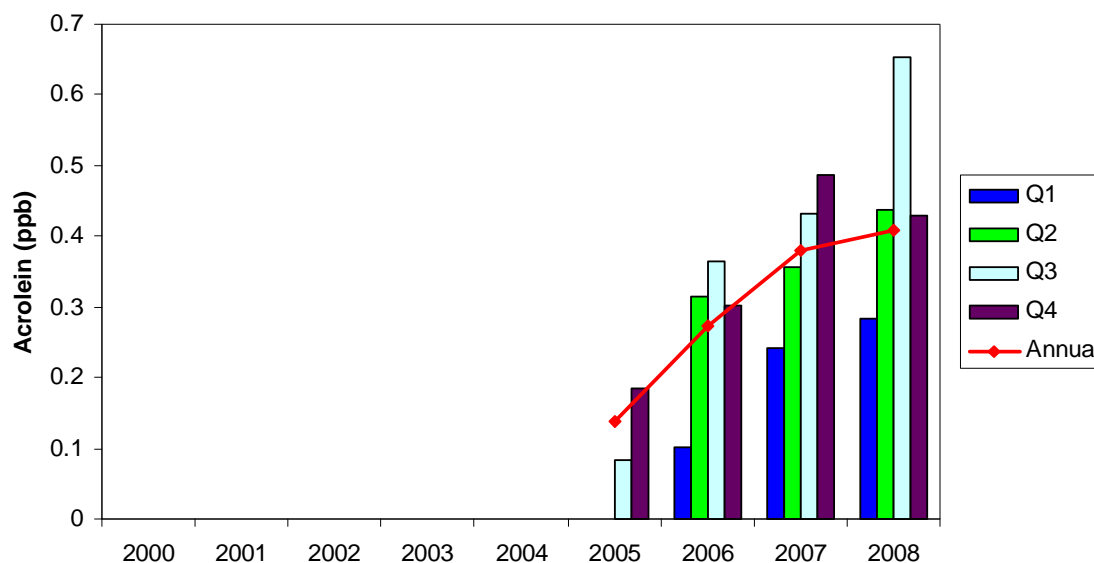
Just under 15 percent of the measured acrolein concentrations are below the MDL, but as with 1,3-butadiene, the percentage of data above the MDL increased in the last few years due to lower reported MDLs. For example, there are only three values below the MDL across all sites for 2007-2009. For years with data for all four quarters (2006-2008), there is an upward trend in annual average measurements at all four sites.

There also appears to be a seasonal difference in acrolein measurements, which may be an artifact of the data quality issue, or may be a true reflection of ambient variation. Quarter 1 has the lowest concentration the most often, and the third quarter has the highest concentration the most often. In Figure 3-72, summer acrolein levels typically exceeded winter concentrations for the same year. Ambient acrolein concentrations may increase by direct emissions or via photochemical production from 1,3-dienes. The chemical production of acrolein will tend to be more significant in warmer months, and this production likely accounts for the seasonal variation in ambient acrolein concentrations illustrated in Figure 3-72.

The relation of acrolein concentrations across the four measurement sites is shown in Figure 3-73. If restricted to 2006-2008, the years for which there were data for

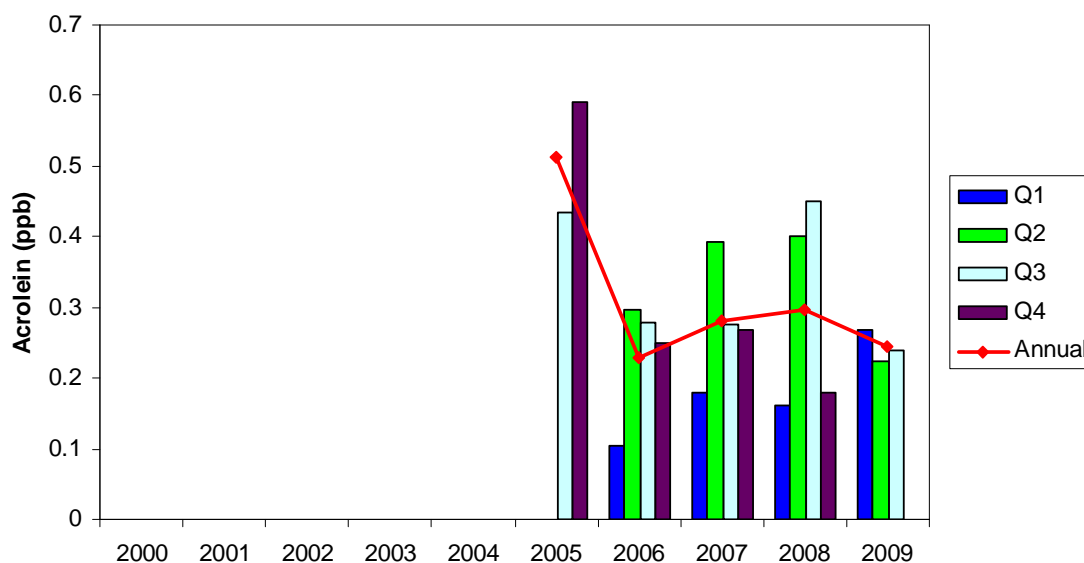
all quarters, Camden and Elizabeth had the highest concentrations of all the sites. In 2006 and 2007, Camden had a slightly higher annual average concentration, and in 2008, Elizabeth had a considerably larger annual average acrolein concentration. New Brunswick had the lowest acrolein concentration of all the sites for 2006-2008.

Figure 3-68. Quarterly and annual average acrolein concentrations for 2005-2008 at Camden



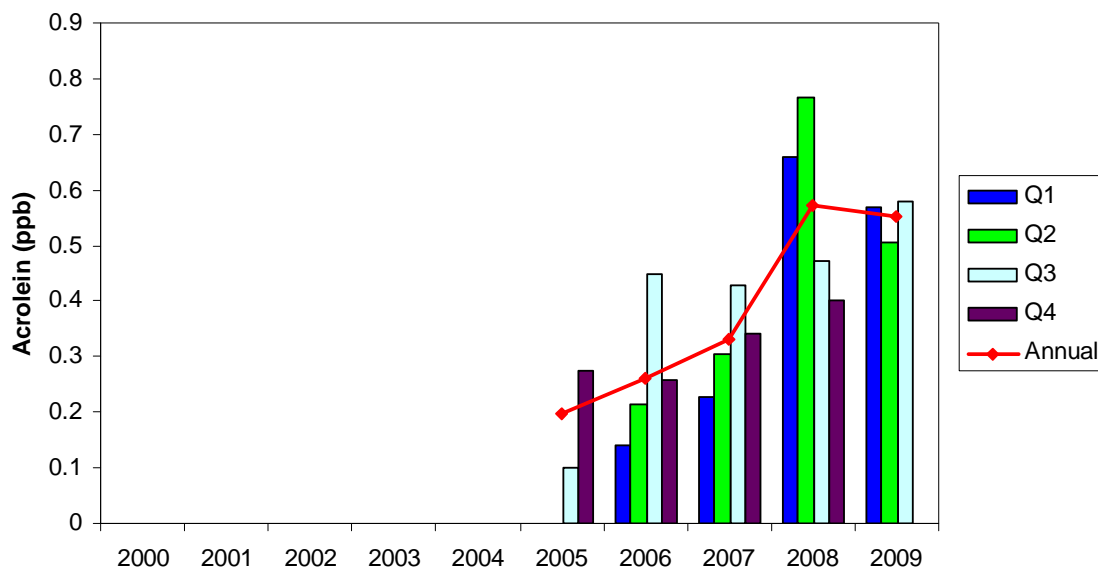
Note: Data were not available for 2009 or the last quarter of 2003 at Camden.

Figure 3-69. Quarterly and annual average acrolein concentrations for 2005-2009 at Chester



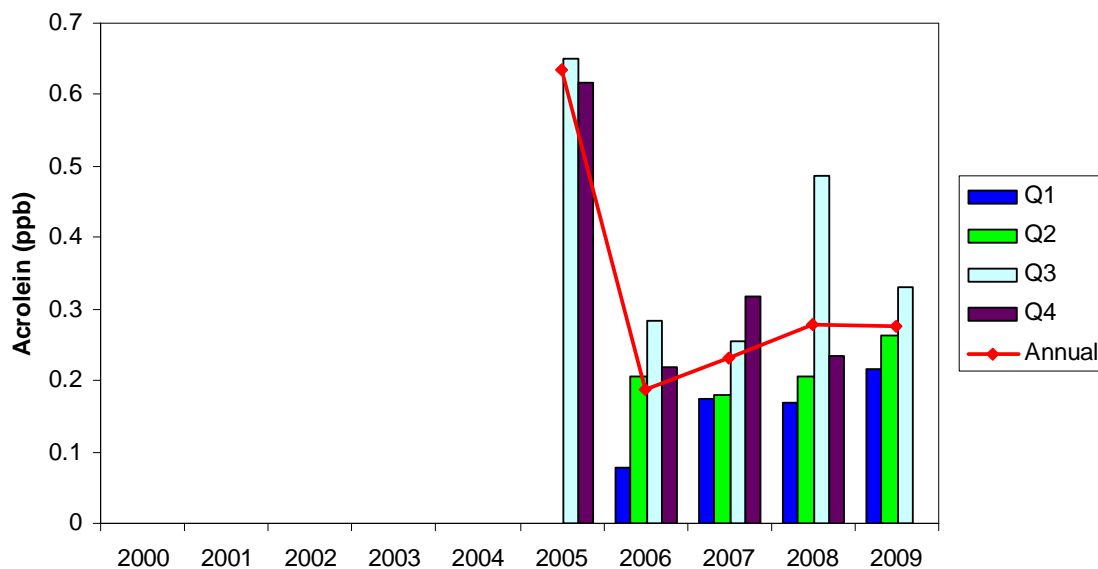
Note: At the time of retrieval, data were not available for the last quarter of 2009 at Chester. Chester came online in 2001.

Figure 3-70. Quarterly and annual average acrolein concentrations for 2005-2009 at Elizabeth



Note: At the time of retrieval, data were not available for the last quarter of 2009 at Elizabeth.

Figure 3-71. Quarterly and annual average acrolein concentrations for 2005-2009 at New Brunswick



Note: At the time of retrieval, data were not available for the last quarter of 2009 at New Brunswick.

Figure 3-72. Summer and winter average acrolein concentrations at Camden, Chester, Elizabeth, and New Brunswick

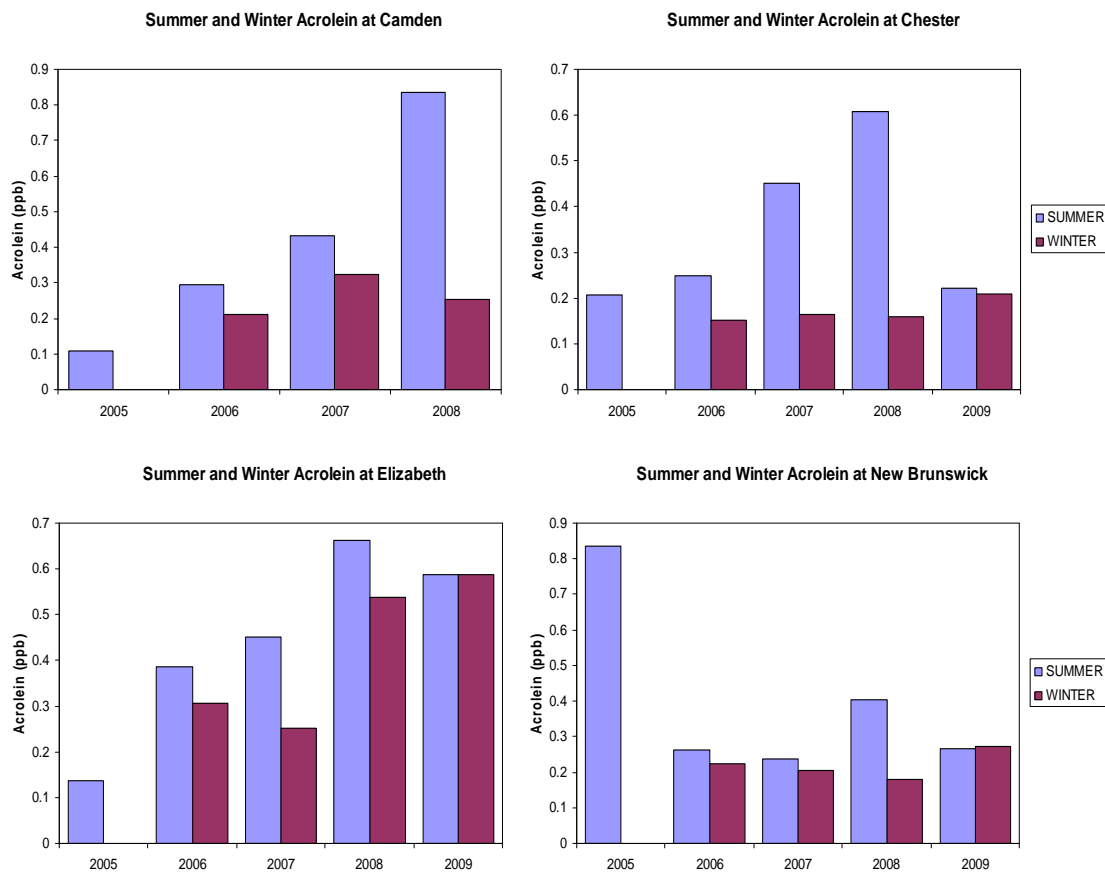
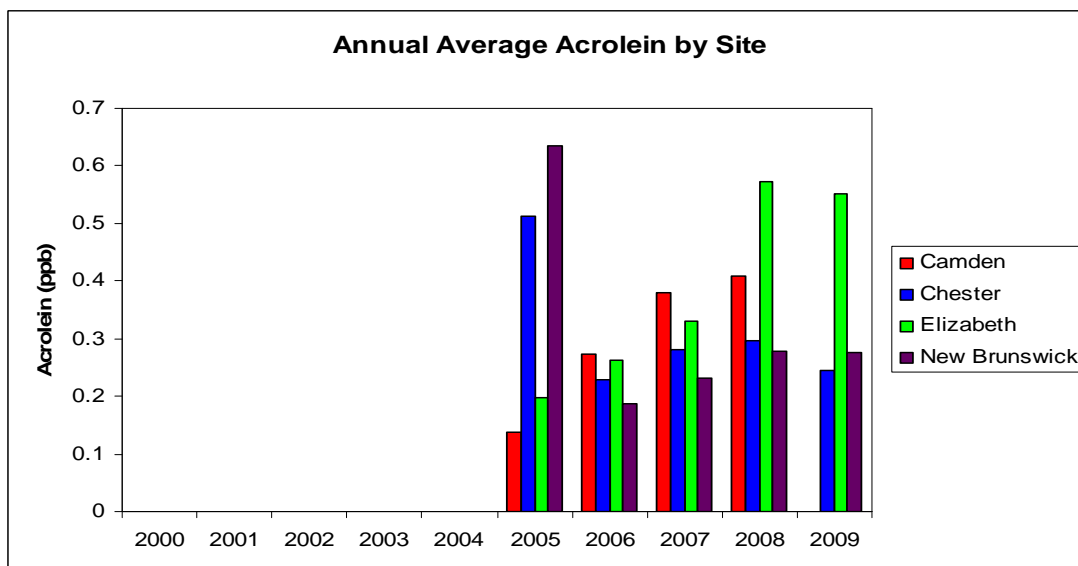


Figure 3-73. Comparison of annual average acrolein concentrations by site



4. PHOTOCHEMICAL AIR TOXICS MODEL EVALUATION

While ambient measurements of air toxics can provide useful information on air toxics levels in the atmosphere and potential community impacts, air quality monitoring is subject to a number of constraints. Monitoring requires a significant investment of resources and, as a result, is often limited in spatial and temporal coverage. In the absence of spatially dense long-term monitoring, air quality modeling can play a vital role in community-scale air toxics assessments. Before they can be used with confidence in such applications, however, models must be evaluated and their limitations addressed to assess their ability to reproduce existing conditions and to determine their efficacy for testing potential policies and scenarios.

NESCAUM and other modeling centers in the region previously performed Community Multi-scale Air Quality Modeling System (CMAQ) simulations over the eastern United States for 2002, 2009, and 2018 to support regional haze evaluations (see NESCAUM, 2006). The CMAQ platform is a state-of-the-science “one-atmosphere” system that treats major atmospheric and land processes (e.g., advection, diffusion, gas phase chemistry, gas-particle mass transfer, nucleation, coagulation, wet and dry deposition, aqueous phase chemistry, etc.) and a range of species (e.g., anthropogenic and biogenic, primary and secondary, gaseous and particulate) in a comprehensive framework. CMAQ has been extensively peer-reviewed, is well-documented, and is regularly updated to reflect the latest changes in scientific understanding. CMAQ has been applied successfully in a range of environments and on many spatial and temporal scales.

For the present analysis, NESCAUM built upon the previous modeling platform used in a regional haze evaluation (NESCAUM, 2006), and launched a 2002 simulation of a more recent version of CMAQ, version 4.7.1, that includes a new chemical mechanism capturing the chemistry of key air toxics species, “cb05txhg.” Even when building upon an existing modeling platform, significant effort is required to develop the necessary modeling inputs (e.g., emissions, meteorology, initial and boundary conditions, photolysis rates, etc.) and parameters (e.g., vertical and horizontal extent of the modeling domain, grid resolution, simulation length, etc.) for a new model run.

This section describes NESCAUM’s use of CMAQ for this project. Section 4.1 provides an overview of air quality modeling for air toxics. Observational data available for comparison with model results are discussed in Section 4.2, and the model evaluation methodology is discussed in Section 4.3. Model specifications for the current CMAQ modeling are presented in Section 4.4. The results of the model performance evaluation for the regional haze modeling and the present modeling are presented in Section 4.5. We discuss these results in Section 4.6.

4.1. Modeling air toxics

Some toxic species are inert, while others are highly reactive. They are emitted from a range of sources, are composed of primary and secondary species, and often have both significant regional and local components. These characteristics make air toxics

particularly challenging to model. Significant local sources of air toxics in some areas may lead to locally elevated levels (“hot spots”) above the urban and regional background. The ability to capture and characterize maximum short-term concentrations of air toxics in addition to long-term averages for national, regional, urban, and community or local-scale areas is necessary to estimate acute and chronic exposures for health risk assessments. Monitoring for air toxics in general is spatially sparse, of low temporal resolution, and limited primarily to urban areas. As a result, models are important tools to provide the temporal and spatial information that cannot be ascertained from observations (Touma et al., 2006).

4.1.1. Types of models: strengths and weaknesses

Different types of models can be used to assess ambient levels of air toxics on wide chemical and spatial scales, determine the importance of emissions sources, and estimate the impacts of emissions control strategies. Historically, Gaussian dispersion models have been used in local assessments of air toxics. These models are limited in that they often do not include a rigorous description of chemistry or biogenic emissions, in some cases do not include non-steady-state meteorology, and are valid only a short distance from the source (less than 50 km) (Touma et al., 2006). Regional background concentrations of air toxics may be significant in some areas, however, and thus long range transport should be considered in urban toxics assessments (Seigneur et al., 2003).

In an annual simulation by Luecken et al. (2006), it was determined that photochemistry accounts for most ambient formaldehyde and acetaldehyde and also significantly influences acrolein. Because these species cannot be accurately modeled without taking chemistry into account, and because they also account for significant health effects (see Section 2), an assessment that incorporates chemical transformations of air toxics will be highly valuable to any assessment of risk. Gridded chemical transport models (CTMs), like CMAQ, simulate the spatiotemporal evolution of many species in a “one-atmosphere” approach with complex chemical mechanisms, emissions from biogenic and anthropogenic sources, and complex meteorology at a range of spatial scales. One major constraint of CTMs is the resolution imposed by its grid cell size. The model has difficulty resolving strong gradients very close to emissions sources and so may not always be able to capture the maximum short-term concentrations responsible for acute exposures. Recent improvements in computational power and model representation at finer scales are allowing for relatively more reliable modeling at resolutions as fine as 1 kilometer (km).

While the effects of subgrid variability will be lessened by modeling increasingly smaller cells, model physics limit current CTM grid cells to sizes no less than 1 km (Isakov et al., 2007). Even at 1 km resolution, there is subgrid variability (SGV) lost in the average values for the cell, and incorporating a description of the SGV with gridded model outputs might be necessary to identify hot spots (Ching et al., 2006b). The characterization of SGV for gridded models is an active area of research, and there are currently a number of approaches to capture both regional and local contributions in air toxics modeling. One approach includes combining a probabilistic description of the subgrid spatial variability with regional modeling concentrations using information from finer-scale modeling studies or coupled large-eddy simulation and photochemistry

models (Ching et al., 2006a; Isakov et al., 2007). Additionally, the results of separate regional chemical transport model and local dispersion model simulations can be combined while taking care to avoid double counting of emissions (Stein et al., 2007; Isakov et al., 2007; Isakov and Venkatram, 2006). Though increasingly computationally-intensive for each additional source, integrated plume-in-grid modeling within a chemical transport model can be activated for select sources. (Plume-in-grid modeling is no longer available in CMAQ as of version 4.7.) Plume-in-grid modeling allows the urban background and local source impacts to be simulated simultaneously while avoiding the concerns of double-counting (Karamchandani et al., 2009). Many air toxics modeling studies employing these techniques have been applied to urban areas around the country, including Houston, Philadelphia, southern California, and New York City (Stein et al., 2007; Ching et al., 2006b; Isakov et al., 2007; Isakov and Venkatram, 2006; Karamchandani et al., 2009). While techniques for estimating and applying SGV are still being developed, a representation of SGV could ultimately be used in conjunction with modeled grid concentrations to offer a distribution of grid values for comparison with point measurements rather than just a single value for model evaluation (Ching et al., 2006b).

While a CTM may not be able to capture maximum concentrations close to a strong source, the ability of a grid model to capture average temporal and spatial trends is valuable, especially for species that have strong regional components or are chemically reactive. In this analysis, NESCAUM generally followed the recommended model evaluation guidelines for fine particulate matter and ozone to guide our statistical and graphical analyses (US EPA, 2007). In doing so, we will refrain from setting strict model performance goals for air toxics at this time due to the fact that the observations from a limited monitoring network may not well represent the volume-average concentrations simulated by the model.

4.1.2. Model uncertainty and sensitivity

There are many independent, potential sources of uncertainty and model bias. Model biases can come from errors in air toxics emissions inventories, inventories of other species involved in chemistry leading to the formation of air toxics (e.g., biogenics), meteorology, chemistry, boundary conditions, and incorrect or incomplete parameterizations of physical and chemical processes. Gridded CTMs are subject to some numerical diffusion from advection routines and artificial dilution in grid cell volumes. Sensitivities to inputs and parameterizations can be highly variable and depend upon many factors, such as chemical species, time of day and year, location, grid resolution, temporal averaging, and more.

There have been many studies to date examining the sensitivity of model outputs to inputs and parameterizations (e.g., Appel et al., 2007; Appel et al., 2008; Appel et al., 2009). NESCAUM did not perform sensitivity analysis on the model to identify highly sensitive parameters. Such an analysis would provide insight and suggest future improvements to model parameterization and inputs.

4.2. Observational data

The temporal and spatial variability of different species may be considerable (Spicer et al., 1996; Luecken et al., 2006). McCarthy et al. (2007) examined the temporal variability of a number of air toxics across the United States, and found some distinct seasonal and diurnal behaviors for different groups of air toxics. Species like 1,3-butadiene and benzene show a diurnal pattern heavily influenced by mobile source emissions having a “morning peak.” The analysis of ambient data in New Jersey and the broader region presented in Section 3 is consistent with this national finding. Formaldehyde shows a daytime peak influenced by photochemical production, which may account for the majority of ambient formaldehyde (Luecken et al., 2006; McCarthy et al., 2007). Acetaldehyde shows a daytime and morning peak because it is heavily influenced by photochemical production in addition to mobile sources (McCarthy et al., 2007). Air toxics measurements are typically in the form of 24-hour averages. While these cannot be used to determine diurnal behavior, PAMS stations measure some air toxics of interest at a higher temporal resolution (one- or three-hour samples, depending on species and season), and these may be used in conjunction with 24-hour average air toxics measurements to examine diurnal variation (Sistla and Aleksic, 2007).

The toxics considered in this analysis vary considerably in their atmospheric behavior, as described in Sections 2 and 3. Formaldehyde and acetaldehyde are emitted directly and are also heavily influenced by photochemistry. Benzene is not produced in the atmosphere but is depleted by reactions with the hydroxyl radical, whose concentrations vary by season and location and may not be very well characterized in CMAQ for all locations and time periods. Benzene is not very reactive and remains in the atmosphere long enough to be transported considerable distances from its source. Conversely, 1,3-butadiene has a much shorter atmospheric lifetime. Benzene and 1,3-butadiene are far more heavily influenced by direct emissions than formaldehyde and acetaldehyde. Acrolein is also significantly impacted by chemistry (e.g., production from species like 1,3-butadiene and destruction by the hydroxyl radical) (Luecken et al., 2006). The different physicochemical behaviors of these air toxic species in conjunction with the spatial and temporal differences in meteorology and emissions suggest that the model will likely have non-uniform levels of success simulating the evolution of each species.

For the model evaluation described below, NESCAUM used 24-hour measurements from NJ Air Toxics sites and New Jersey PAMS sites only (see Section 3.1). The data were processed into CASTNET-like format for use with model evaluation software using NESCAUM-generated Python 3.x scripts.

4.3. Model evaluation methodology and discussion

Model results and measurements are not always directly comparable, as model results represent the average concentration over a grid cell volume, while observations are measured at a single point. Due to this “change of support” problem, even in the case of perfect model performance, one cannot expect modeled concentrations, which are volume-averages, to match the observed concentrations that are point values (Gelfand et al., 2000; Ching et al., 2006a). As Ching et al. (2006a) put it, “any observation reflects an event out of a population, while model predictions represent an average of the population.” As a result, in some cases, comparisons between modeled and observed

concentrations may not effectively address how well the model simulates the physicochemical behavior of pollutants (Park et al., 2006). When modeled concentrations are compared with observations, one should consider whether the measurements are representative of a wider area or if the monitor area is characterized by strong spatial concentration gradients (McNair et al., 1996). Indeed, for certain species and locations, monitors may be sited such that their measurements may approximate grid cell average concentrations. Air toxics monitors are usually sited in urban areas, and the concentration gradients around monitors may differ for each species and location. Regional sources can be the most significant contributors to toxics concentrations at a monitor, while in other locations or for other species, local sources may dominate. Spicer et al. (1996) found that if averaging times are long enough to smooth out the temporal patterns, measurements made in one neighborhood in some cities may be representative of most of the urban area. Conversely, Luecken et al. (2006) found in some cases, a factor of two to three difference in measurements from monitors within one model grid cell. In a study of VOC spatial variation in Camden, it was found that, for a number of short measurement campaigns, the spatial distributions in the neighborhood including the Camden air toxics monitor were relatively uniform compared to measurements at a nearby hot spot (Zhu et al., 2008).

If an area contains a few concentrated sources of a species rather than a more homogeneous spread of sources throughout the area, significant spatial concentration gradients may exist. Toxics “hot spots” may result in areas that are not well-characterized by the volume-average modeled concentrations. Monitors may be near a major point source or roadway or in an area with complex microscale meteorology that may not be resolved by the grid-based model. Modeled concentrations may better capture local effects as grid resolution is increased, but some fast, local processes may not be well represented in a grid model even at the limits of grid cell size. NESCAUM’s goal for this project is not necessarily to develop the model so that its concentrations match observations as closely as possible, but rather to evaluate the model in its current state with these limitations in mind and to characterize the extent of agreement between model and observational data.

Due to the strong seasonal variations for some species, special care needs to be taken when generating the average values that will be used for comparisons with model outputs. McCarthy et al. (2007) found that for some pollutants, concentrations may vary by season by a factor of two, and that such differences could significantly bias annual average concentrations if more measurements are taken in one season than another. Formaldehyde and 1,3-butadiene have very strong seasonal variability, with 1,3-butadiene peaking in colder months while the opposite is true for formaldehyde. When calculating averages, NESCAUM matched model results with observations in space and time to assess the day-by-day and monthly performance of the model, in addition to producing summary statistics.

Models have more difficulty accurately simulating very low concentrations as the model signal to noise ratio becomes smaller. While we employ statistical measures to quantify the model evaluation, in cases when a large percentage of measurements are below the MDL for a species, the model evaluation becomes more qualitative in nature. When developing metrics and goals for model performance for particulate matter, Boylan

and Russell (2006) suggested using less stringent model performance goals for lower concentrations, as these concentrations would be more difficult to simulate and possibly less important from a policy standpoint.

There are no standard metrics or performance goals for air toxics model evaluation, but a number of statistics have been evaluated for use in model performance evaluations for other species (Yu et al., 2006; Boylan and Russell, 2006). For 24-hour and monthly average concentrations, NESCAUM calculated normalized mean bias (NMB) and normalized mean error (NME) for comparison with Luecken et al. (2006); and mean fractional error (MFE) and mean fractional bias (MFB), as recommended by Boylan and Russell (2006) for performance evaluations that incorporate both observations and predictions in the normalization. MFE and MFB are “symmetric” metrics, contrary to NME and NMB which give more weight to model overpredictions than underpredictions (Boylan et al., 2006; Yu et al., 2006). Past performance indicates that it is not unusual for model predictions to fall within a factor of two of observations (Luecken et al., 2006; Touma et al., 2006). In a past study for a 2001 simulation, modeled and observed annual averages of 1,3-butadiene, formaldehyde, acetaldehyde, and benzene were compared for the Camden site (Ching et al., 2004). Ratios of modeled (at 4 km resolution) to measured mean concentrations for 1,3-butadiene, formaldehyde, acetaldehyde, and benzene were 0.55, 0.79, 1.19, and 0.92, respectively. These were somewhat better results than those for a 36 km resolution model simulation.

Luecken et al. (2006) evaluated formaldehyde, acetaldehyde, 1,3-butadiene, benzene, and acrolein concentrations from an annual simulation of CMAQ version 4.4. The model was run over the continental U.S. for the year 2001 with a grid cell resolution of 36 km×36 km. They compared model results for the above species (minus acrolein due to the lack of valid measurements) to observations at 35 monitors in eight cities. In general, though the model had a tendency to underpredict concentrations, it reproduced temporal behavior. The majority of model predictions were within a factor of two of the observations with the exception of 1,3-butadiene, where only 46 percent of the modeled monthly-average concentrations fell within a factor of two of observations. They noted that comparisons with 1,3-butadiene were made difficult by the low concentrations and the “potential analysis difficulties due to elution of 1,3-butadiene on the tail of acetaldehyde” (Luecken et al., 2006). We note that this study relied upon monitoring data that are highly uncertain, given the difficulties in monitoring acrolein as discussed in Section 3.3.

We present the model performance evaluation results benchmarked against the Luecken et al. (2006) study. While the smaller grid cell size of 12 km used in our analysis should allow the model to capture smaller scale variations, the model will still likely have difficulties capturing observed concentrations for areas and species with strong concentration gradients around the monitors. Temporal averaging may minimize the impacts of different causes of some subgrid spatial and temporal variability (Spicer et al., 1996). Section 4.5 presents the results of our model performance evaluations.

Statistics offers a quantitative evaluation of model performance, but graphical analysis was also performed. We generated box plots and scatter plots to elucidate toxics’ temporal and spatial characteristics and also indicated the location of any obvious errors or anomalies. Plots of temporal patterns help indicate whether there is any time of

day, day of the week, or season when model performance suffers. Scatter plots may indicate sections of the concentration distribution (e.g., very high or very low concentrations) where the model does not perform well (US EPA, 2007). These plots in conjunction with standard statistical measures will form the foundation for the evaluation of CMAQ version 4.7.1 with air toxics for the northeast U.S. with a focus on New Jersey monitoring sites.

The discussion above highlights the uncertainties and complexities of comparing observed values for air toxics against model predictions. Furthermore, the model chemistry for the original modeling (the CB4 chemical mechanism without explicit representation of individual air toxics) does not match that of the new modeling (the

Table 4-1. Mapping CB05 species to CB4 species

CB05			CB4		
Species	Description	Carbons	Species	Description	Carbons
ALD2	Acetaldehyde	2	ALD2	Acetaldehyde and higher aldehydes	2
ALDX	Prionaldehyde and higher aldehydes	2			
ETH	Ethene	2	ETH	Ethene	2
FORM	Formaldehyde	1	FORM	Formaldehyde	1
ISOP	Isoprene	5	ISOP	Isoprene	5
TERP	Terpene	10			
OLE	Terminal olefin carbon bond (R-C=C)	2	OLE	Olefin carbon bond (R-C=C)	2
IOLE	Internal olefin carbon bond (R-C=C-R)	4			
PAR	Paraffin carbon bond (C-C)	1	PAR	Paraffin carbon bond (C-C)	1
ETHA	Ethane	2			
TOL	Toluene and other monoalkyl aromatics	7	TOL	Toluene and other monoalkyl aromatics	7
XYL	Xylenes and other polyalkyl aromatics	8	XYL	Xylenes and other polyalkyl aromatics	8

The CB05 chemical mechanism (Yarwood et al., 2005) provides more explicit representation of reactive oxidative species as compared to the CB4 chemical mechanism (Gery et al., 1989). This mapping scheme between CB4 species and CB05 species is restricted to model species that are relevant to air toxics observations.

Note: CB4 species MEOH (methanol) and ETOH (ethanol) are not represented here because no observations were available for these species.

Source: Yarwood et al. (2005).

CB05 chemical mechanism with explicit toxics representation for a variety of species). Comparing the two models' individual performance is complicated by the differences in their representation of air toxics species. However, observations of individual air toxics can be aggregated into model species for direct comparison with predicted concentrations using model chemistry. CB05 predictions can also be easily converted into CB4 lumped species, as they are less refined. For instance, the sum of CB05 species ALD2 (acetaldehyde) and ALDX (higher aldehydes) is equivalent to the CB4 species ALD2 (acetaldehyde and higher aldehydes). The mapping of CB05 lumped model species into CB4 model species is presented in Table 4-1. Observations of reactive organic compounds (not necessarily air toxics) were translated into model species through use of the emitdb.xls workbook (Carter, 2011). Therefore, by assessing each model's performance against observations lumped into CB4 species, both models are evaluated on an equivalent basis.

NESCAUM used the Atmospheric Model Evaluation Tool (AMET) version 1.1 (Gilliam et al., 2005) to generate plots and statistics for this model evaluation. Because AMET is hard-coded to evaluate only a certain set of meteorological and chemical data, NESCAUM adjusted some of the key R, Perl, and csh scripts so that it would accommodate air toxics comparison data. Statistical results from AMET were additionally processed using custom Python 3.x scripts to develop summaries by site and time.

4.4. CMAQ model settings

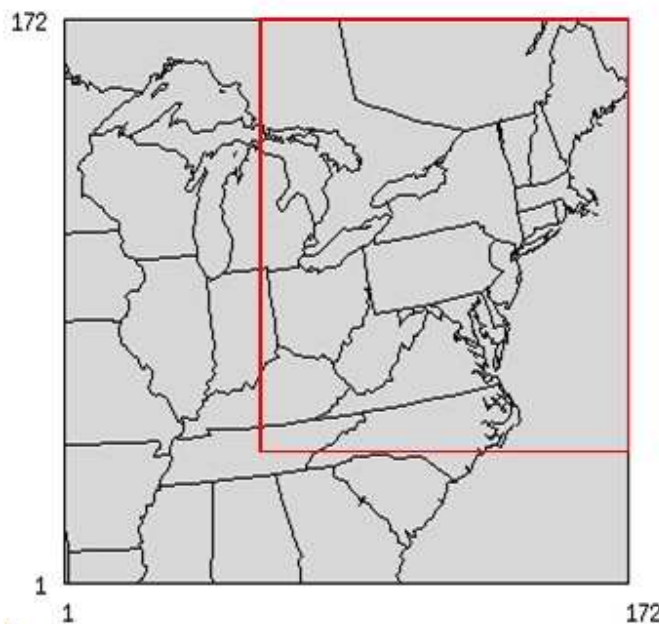
This section presents key settings used in modeling air toxics in New Jersey using the CMAQ version 4.7.1 modeling system. Differences between the regional haze modeling and current modeling are highlighted. The model domain is discussed in Section 4.4.1, meteorology in Section 4.4.2, emissions in Section 4.4.3, and boundary and initial conditions in Section 4.4.4.

4.4.1. Domain

The domain used in our previous simulations was a 172×172 lateral cell 12 km resolution domain over the eastern United States using a Lambert Conformal Conic (LCC) projection with parallels at 33°N and 45°N. There were 22 model layers, spanning from the ground to 50 millibar (mb). For the new simulation with CMAQ version 4.7.1 with air toxics, a new domain has been developed in light of the intensive computational requirements necessary for a long term simulation and this new chemical mechanism. This new domain, marked with a red boundary, is shown in relation to the regional haze domain in Figure 4-1. It shares the same vertical structure, resolution, and north and east boundaries as the previous domain but is nearly halved in size. The new domain contains 112×132 lateral cells. The NOAA HYSPLIT model was used to choose a western boundary that was about two days' transport distance from New Jersey.

4.4.2. Meteorology

A non-hydrostatic MM5 simulation for 2002 was prepared by the University of Maryland for the regional haze CMAQ simulations. These MM5 outputs were used to generate meteorological inputs for the present CMAQ simulation. While meteorological

Figure 4-1. CMAQ modeling domains

Note: The smaller domain, indicated in red, was used for the annual simulation of CMAQ version 4.7.1 with toxics. The larger domain was used in previous CMAQ simulations for regional haze and will be used for a subset of days to test the sensitivity of species' concentrations to domain size.

inputs had been previously generated for CMAQ, these inputs needed to be reprocessed using a newer version of the Meteorology-Chemistry Interface Processor (MCIP v. 3.4.1) in order to generate the additional meteorological variables required in CMAQ version 4.7.1.

4.4.3. Emissions

National Emissions Inventory (NEI) files for toxic and criteria pollutants for 2002 were obtained from the US EPA. The Sparse Matrix Operator Kernel Emissions (SMOKE) model was used to develop model-ready emissions files for January and July. These months are used as representative of winter and summer conditions, respectively.

4.4.4. Boundary conditions and initial conditions

NESCAUM worked closely with Deborah Luecken of the US EPA's Atmospheric Modeling and Analysis Division to develop boundary conditions for our simulation. Boundary conditions were derived from a combination of two simulations run at 12 km resolution over the eastern half of the United States. Most species were derived from an annual CMAQ version 4.6 CB4 with Toxics simulation. Because there were significant updates to the aerosol module between CMAQ versions 4.6 and 4.7, organic aerosol and semi-volatile species were taken from a separate CMAQ version 4.7 simulation (not run with the toxics mechanism). NESCAUM also obtained the necessary boundary files and received an updated acrolein yield that improved upon the yield in the default chemical mechanism in the model. The model was run on a five-node dual-processor Linux cluster. The January simulation included a three-day spin-up period, and the July

simulation a 14-day spin-up period to reduce the impact of clean initial condition fields on the model results.

4.5. Model evaluation results

This section presents the results of the performance model evaluations for both the regional haze CMAQ model (labeled NJ_haze_2002) and new CMAQ with air toxics model (labeled NJ_tox_2002). A summary of comparable statistics from these two model evaluations is presented in Table 4-2, and detailed results for each of the models are presented in Sections 4.5.1 and 4.5.2, including monthly box plots and scatter plots for all available months using data from the NJ Air Toxics network. Charts using data from the PAMS network are also presented when available for diurnal profiles, daily scatter plots, and monthly plots similar to those for the NJ Air Toxics network.

According to the summary-level statistics for CB4 species shown in Table 4-2, model performance for air toxics is generally poor, with metrics varying significantly by month, species, and model. With a few exceptions, these results are generally biased high for both models, and the NJ_haze_2002 model shows somewhat lower levels of NMB and NME, but slightly higher levels of MFB and MFE.

Both models grossly overpredict paraffin bond VOCs by a factor of 10 or more in the summer; NJ_haze_2002 outperforms NJ_tox_2002 in the winter for paraffins, though the error is still seven-fold. Both models do better in their average monthly predictions for olefins, which are highly reactive. The NJ_haze_2002 predictions for olefins are more accurate in the winter, with both models moderately overpredicting, while NJ_tox_2002 has lower error in the summer, when both models moderately underpredict. Monthly average predictions of formaldehyde are generally biased low within a factor of two (except for winter MNB for NJ_haze_2002) and both models have reasonable performance. Higher aldehydes are biased high in both models in both summer and winter, though NJ_haze_2002 does slightly better, especially in winter. Monthly average predictions for higher aldehydes are generally within a factor of two, except for NMB in the summer, when predictions of both models are slightly higher. Predictions of toluene are biased slightly higher in both models and in both summer and winter, but generally match well against observations, with NJ_haze_2002 performing better in the winter and NJ_tox_2002 better in the summer. Both models performed reasonably well for xylenes, except for NJ_haze_2002 in the winter, which showed a 4-fold overprediction. Summer monthly-average predictions of xylenes were within 15 percent for NJ_tox_2002.

The overpredictions may be due to the inherent inadequacies of comparing model species against incomplete measurements of monitored species. For instance, some species of paraffins may have gone undetected at the monitors. Direct comparison of specific species for the NJ_tox_2002 model may provide additional insight into the performance of the model for paraffins and olefins. Other sensitivity analyses will provide insight into how to improve model performance.

Table 4-2. Domain-wide summary of model performance evaluation results against 2002 NJ Air Toxics data

Month	CB4 species	Model	N	Obs. mean	Mod. mean	%NMB	%NME	%MFB	%MFE
January	ALD2	NJ_haze_2002	18	0.862	1.269	47.25	59.48	36.0	46.5
January	ALD2	NJ_tox_2002	18	0.862	1.670	93.76	93.76	61.3	61.3
January	FORM	NJ_haze_2002	18	1.798	0.934	-48.06	48.25	-58.3	58.6
January	FORM	NJ_tox_2002	18	1.798	1.399	-22.19	39.34	-22.9	42.7
January	OLE	NJ_haze_2002	18	1.577	1.822	15.56	93.95	70.1	89.2
January	OLE	NJ_tox_2002	18	1.577	2.610	65.56	130.90	94.9	107.9
January	PAR	NJ_haze_2002	18	8.284	71.863	767.44	767.44	158.7	158.7
January	PAR	NJ_tox_2002	18	8.284	96.384	1063.44	1063.44	167.8	167.8
January	TOL	NJ_haze_2002	18	2.463	2.581	4.80	85.93	36.6	72.4
January	TOL	NJ_tox_2002	18	2.463	3.069	24.60	105.89	51.0	85.7
January	XYL	NJ_haze_2002	18	1.306	4.425	238.78	238.78	108.8	108.8
January	XYL	NJ_tox_2002	18	1.306	1.935	48.15	83.40	38.0	63.9
July	ALD2	NJ_haze_2002	16	1.516	3.054	101.48	105.43	72.2	75.1
July	ALD2	NJ_tox_2002	13	1.595	3.772	136.53	136.53	83.5	83.5
July	FORM	NJ_haze_2002	16	4.511	4.434	-1.72	43.23	11.5	46.6
July	FORM	NJ_tox_2002	13	4.783	3.533	-26.14	40.79	-15.9	46.7
July	OLE	NJ_haze_2002	16	2.232	2.198	-1.53	85.98	50.0	80.1
July	OLE	NJ_tox_2002	13	2.388	1.751	-26.68	77.07	30.6	70.2
July	PAR	NJ_haze_2002	16	7.504	84.397	1024.64	1024.64	164.7	164.7
July	PAR	NJ_tox_2002	13	8.032	92.925	1057.01	1057.01	165.7	165.7
July	TOL	NJ_haze_2002	16	1.377	2.314	68.02	110.00	47.4	74.8
July	TOL	NJ_tox_2002	13	1.552	1.808	16.53	77.63	9.9	62.0
July	XYL	NJ_haze_2002	16	0.946	1.457	54.06	103.25	37.9	75.0
July	XYL	NJ_tox_2002	13	1.079	1.242	15.05	77.07	8.4	66.5

NJ_haze_2002 = regional haze model with CB4 chemistry; NJ_tox_2002 = NJ air toxics model with CB05 chemistry. NMB = Normalized mean bias. NME = Normalized mean error. MFB = Mean fractional bias. MFE = Mean fractional error.

Note: Slight differences in observational data values are the result of differences in model settings for start- and end-time. NJ_haze_2002 was set with 0:00 GMT as the start/end time; NJ_tox_2002 was set with 0:00 eastern as the start/end time.

4.5.1. Regional haze model evaluation results

This section presents results of the model evaluation from the regional haze model (labeled NJ_haze_2002). Results are presented in the form of summary statistics for domain-wide (all monitors) and specific monitors, as well as in box plots, which help characterize the model versus observed temporal patterns, and scatter plots that show accuracy and precision of the model predictions.

Statistics

Domain-wide results

Table 4-3. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for January

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
January	PAR	NJ_haze_2002	18	8.284	71.863	767.44	767.44	158.7	158.7
January	OLE	NJ_haze_2002	18	1.577	1.822	15.56	93.95	70.1	89.2
January	ALD2	NJ_haze_2002	18	0.862	1.269	47.25	59.48	36	46.5
January	TOL	NJ_haze_2002	18	2.463	2.581	4.8	85.93	36.6	72.4
January	XYL	NJ_haze_2002	18	1.306	4.425	238.78	238.78	108.8	108.8
January	FORM	NJ_haze_2002	18	1.798	0.934	-48.06	48.25	-58.3	58.6

Table 4-4. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for February

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
February	PAR	NJ_haze_2002	19	12.63	84.98	572.93	572.93	148.70	148.70
February	OLE	NJ_haze_2002	19	1.85	1.99	7.66	57.71	41.10	63.70
February	ALD2	NJ_haze_2002	19	1.12	1.43	27.74	47.76	26.60	41.20
February	TOL	NJ_haze_2002	19	1.72	3.21	86.83	95.39	53.60	61.20
February	XYL	NJ_haze_2002	19	1.31	4.78	265.23	265.91	110.00	110.40
February	FORM	NJ_haze_2002	19	2.79	1.00	-64.17	64.85	-76.50	78.50

Table 4-5. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for March

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
March	PAR	NJ_haze_2002	18	8.35	60.09	620.12	620.12	148.20	148.20
March	OLE	NJ_haze_2002	18	1.92	1.16	-39.80	85.88	45.30	78.30
March	ALD2	NJ_haze_2002	18	0.95	1.00	5.93	40.76	7.60	39.60
March	TOL	NJ_haze_2002	18	1.18	1.99	68.65	81.17	45.30	59.10
March	XYL	NJ_haze_2002	18	1.19	2.08	75.36	100.34	62.20	78.40
March	FORM	NJ_haze_2002	18	2.46	0.65	-73.56	74.94	-80.40	86.50

Table 4-6. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for April

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
April	PAR	NJ_haze_2002	20	7.65	53.38	598.19	598.19	149.70	149.70
April	OLE	NJ_haze_2002	20	1.09	1.18	7.48	64.74	46.20	66.30
April	ALD2	NJ_haze_2002	18	1.06	1.10	3.92	35.69	3.60	35.00
April	TOL	NJ_haze_2002	20	0.90	1.61	78.38	97.19	55.40	65.90
April	XYL	NJ_haze_2002	20	0.78	1.73	120.76	130.91	74.00	79.50
April	FORM	NJ_haze_2002	18	2.90	1.06	-63.50	63.59	-73.10	73.20

Table 4-7. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for May

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
May	PAR	NJ_haze_2002	21	6.95	59.38	754.00	754.00	155.10	155.10
May	OLE	NJ_haze_2002	20	0.64	1.21	88.78	91.62	70.00	71.80
May	ALD2	NJ_haze_2002	20	1.04	1.21	15.85	44.18	14.90	41.80
May	TOL	NJ_haze_2002	20	0.96	1.84	92.20	131.36	66.10	84.60
May	XYL	NJ_haze_2002	20	0.54	1.63	198.57	203.97	96.10	97.90
May	FORM	NJ_haze_2002	20	3.73	1.29	-65.41	68.24	-62.50	70.80

Table 4-8. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for June

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
June	PAR	NJ_haze_2002	19	7.39	85.32	1054.54	1054.54	164.60	164.60
June	OLE	NJ_haze_2002	19	1.56	1.93	24.26	92.93	62.70	91.60
June	ALD2	NJ_haze_2002	19	1.04	2.47	138.56	145.50	82.70	88.10
June	TOL	NJ_haze_2002	19	1.10	2.71	147.13	155.14	74.90	81.20
June	XYL	NJ_haze_2002	19	0.88	1.64	86.81	112.74	50.60	69.70
June	FORM	NJ_haze_2002	19	3.14	2.99	-5.05	65.60	3.30	68.80

Table 4-9. Domain-wide summary of regional haze model performance evaluation results against 2002 PAMS data for June

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
June	PAR	NJ_haze_2002	2068	29.50	71.00	140.69	171.08	102.60	108.00
June	OLE	NJ_haze_2002	2068	0.58	1.73	198.17	218.02	101.00	107.20
June	ALD2	NJ_haze_2002	1802	0.25	2.48	897.51	902.15	162.60	163.10
June	ISOP	NJ_haze_2002	1905	0.29	1.43	399.28	410.32	90.20	124.70
June	TOL	NJ_haze_2002	2065	0.61	1.71	178.52	199.75	103.60	108.30
June	XYL	NJ_haze_2002	1923	0.36	1.02	183.74	202.62	112.30	116.20
June	ETH	NJ_haze_2002	2063	0.80	1.37	72.42	95.89	66.70	76.30

Table 4-10. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for July

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
July	PAR	NJ_haze_2002	16	7.504	84.397	1024.64	1024.64	164.7	164.7
July	OLE	NJ_haze_2002	16	2.232	2.198	-1.53	85.98	50	80.1
July	ALD2	NJ_haze_2002	16	1.516	3.054	101.48	105.43	72.2	75.1
July	TOL	NJ_haze_2002	16	1.377	2.314	68.02	110	47.4	74.8
July	XYL	NJ_haze_2002	16	0.946	1.457	54.06	103.25	37.9	75
July	FORM	NJ_haze_2002	16	4.511	4.434	-1.72	43.23	11.5	46.6

Table 4-11. Domain-wide summary of regional haze model performance evaluation results against 2002 PAMS data for July

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
July	PAR	NJ_haze_2002	2190	26.73	75.41	182.12	208.02	115.00	118.90
July	OLE	NJ_haze_2002	2086	0.57	2.12	270.76	289.79	115.00	119.20
July	ALD2	NJ_haze_2002	1771	0.26	3.05	1077.99	1085.11	169.50	170.00
July	ISOP	NJ_haze_2002	1859	0.38	1.80	368.48	375.47	87.80	123.00
July	TOL	NJ_haze_2002	2188	0.62	1.73	180.46	208.73	108.30	114.00
July	XYL	NJ_haze_2002	2024	0.32	0.94	193.30	207.65	113.00	116.80
July	ETH	NJ_haze_2002	2012	0.82	1.53	87.41	108.25	73.80	83.00

Table 4-12. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for August

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
August	PAR	NJ_haze_2002	12	7.33	102.41	1297.18	1297.18	175.40	175.40
August	OLE	NJ_haze_2002	12	2.57	2.50	-2.73	100.37	69.10	93.00
August	ALD2	NJ_haze_2002	10	0.99	2.63	165.77	165.77	95.80	95.80
August	TOL	NJ_haze_2002	12	4.63	3.35	-27.72	102.71	35.90	95.40
August	XYL	NJ_haze_2002	12	0.99	2.17	119.02	119.02	68.00	68.00
August	FORM	NJ_haze_2002	10	2.80	2.67	-4.58	28.02	-2.50	39.00

Table 4-13. Domain-wide summary of regional haze model performance evaluation results against 2002 PAMS data for August

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
August	PAR	NJ_haze_2002	2116	26.85	84.11	213.24	223.44	115.30	117.40
August	OLE	NJ_haze_2002	2115	0.58	2.27	293.60	305.43	118.80	122.10
August	ALD2	NJ_haze_2002	1873	0.27	3.17	1058.13	1059.49	168.20	168.30
August	ISOP	NJ_haze_2002	1946	0.41	2.00	390.72	402.91	82.70	125.80
August	TOL	NJ_haze_2002	2115	0.68	2.06	205.57	224.85	114.90	118.30
August	XYL	NJ_haze_2002	1918	0.41	1.17	182.85	196.48	113.10	116.30
August	ETH	NJ_haze_2002	2094	0.90	1.74	93.95	123.36	82.80	91.70

Table 4-14. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for September

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
September	PAR	NJ_haze_2002	19	4.26	77.72	1726.58	1726.58	176.90	176.90
September	OLE	NJ_haze_2002	18	0.68	1.99	191.71	191.71	103.20	103.20
September	ALD2	NJ_haze_2002	19	0.79	2.06	161.82	161.82	89.70	89.70
September	TOL	NJ_haze_2002	18	1.20	2.39	99.78	115.86	64.00	76.10
September	XYL	NJ_haze_2002	18	0.61	2.01	229.47	229.47	102.60	102.60
September	FORM	NJ_haze_2002	19	2.18	2.32	5.98	43.15	11.70	42.60

Table 4-15. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for October

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
October	PAR	NJ_haze_2002	19	5.99	90.51	1411.82	1411.82	174.30	174.30
October	OLE	NJ_haze_2002	18	1.53	2.05	33.51	82.56	60.90	80.70
October	ALD2	NJ_haze_2002	19	0.58	1.67	190.90	200.14	96.00	100.60
October	TOL	NJ_haze_2002	18	1.69	3.35	98.07	101.85	58.50	66.40
October	XYL	NJ_haze_2002	18	0.90	2.94	225.90	226.36	96.50	97.10
October	FORM	NJ_haze_2002	19	1.11	1.06	-4.32	76.14	-6.10	89.20

Table 4-16. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for November

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
November	PAR	NJ_haze_2002	20	6.69	95.41	1325.13	1325.13	173.00	173.00
November	OLE	NJ_haze_2002	20	1.48	2.14	44.32	87.38	69.70	84.30
November	ALD2	NJ_haze_2002	20	0.77	1.46	88.40	103.14	69.80	80.80
November	TOL	NJ_haze_2002	20	1.31	3.64	178.76	178.76	95.70	95.70
November	XYL	NJ_haze_2002	19	0.89	3.77	325.25	325.25	127.70	127.70
November	FORM	NJ_haze_2002	20	1.11	0.68	-38.68	67.50	-23.50	89.90

Table 4-17. Domain-wide summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for December

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
December	PAR	NJ_haze_2002	15	5.84	71.29	1121.19	1121.19	168.50	168.50
December	OLE	NJ_haze_2002	15	1.00	1.76	75.48	87.15	74.20	77.30
December	ALD2	NJ_haze_2002	14	0.69	1.18	69.71	99.96	54.60	77.20
December	TOL	NJ_haze_2002	15	1.23	2.55	106.71	118.91	85.40	87.80
December	XYL	NJ_haze_2002	15	0.64	4.14	551.66	551.66	153.50	153.50
December	FORM	NJ_haze_2002	14	1.01	0.92	-9.03	80.52	4.20	88.30

*Site-specific results***Table 4-18. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for January at Camden Lab**

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Camden	January	PAR	NJ_haze_2002	4	8.36	82.98	893.21	893.21	163.70	163.70
Camden	January	OLE	NJ_haze_2002	4	1.50	2.03	35.79	55.17	50.70	62.60
Camden	January	ALD2	NJ_haze_2002	4	0.84	1.38	64.17	64.17	49.70	49.70
Camden	January	TOL	NJ_haze_2002	4	4.15	2.73	-34.25	97.53	42.40	107.20
Camden	January	XYL	NJ_haze_2002	4	0.86	3.89	353.03	353.03	126.90	126.90
Camden	January	FORM	NJ_haze_2002	4	1.45	1.22	-16.06	17.13	-15.20	16.60

Table 4-19. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for January at Rutgers University

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Rutgers	January	PAR	NJ_haze_2002	5	9.22	69.33	651.58	651.58	153.50	153.50
Rutgers	January	OLE	NJ_haze_2002	5	0.64	1.79	180.88	180.88	98.40	98.40
Rutgers	January	ALD2	NJ_haze_2002	5	0.90	1.26	38.87	45.07	33.90	38.60
Rutgers	January	TOL	NJ_haze_2002	5	3.40	2.37	-30.09	41.93	-15.80	36.70
Rutgers	January	XYL	NJ_haze_2002	5	2.39	4.41	84.46	84.46	58.80	58.80
Rutgers	January	FORM	NJ_haze_2002	5	1.93	0.86	-55.44	55.44	-67.60	67.60

Table 4-20. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for January at Chester

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Chester	January	PAR	NJ_haze_2002	5	4.80	45.68	852.06	852.06	160.70	160.70
Chester	January	OLE	NJ_haze_2002	5	0.32	1.20	278.35	278.35	115.80	115.80
Chester	January	ALD2	NJ_haze_2002	5	0.96	0.87	-9.70	24.06	-9.20	24.20
Chester	January	TOL	NJ_haze_2002	5	1.09	1.46	33.67	67.42	32.30	56.80
Chester	January	XYL	NJ_haze_2002	5	1.01	3.35	231.62	231.62	106.00	106.00
Chester	January	FORM	NJ_haze_2002	5	2.28	0.63	-72.38	72.38	-113.60	113.60

Table 4-21. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for January at Elizabeth

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Elizabeth	January	PAR	NJ_haze_2002	4	11.40	96.64	747.93	747.93	157.40	157.40
Elizabeth	January	OLE	NJ_haze_2002	4	4.41	2.43	-44.84	74.80	-2.90	71.00
Elizabeth	January	ALD2	NJ_haze_2002	4	0.71	1.68	136.62	136.62	81.30	81.30
Elizabeth	January	TOL	NJ_haze_2002	4	1.32	4.09	210.38	210.38	101.60	101.60
Elizabeth	January	XYL	NJ_haze_2002	4	0.77	6.33	722.05	722.05	156.70	156.70
Elizabeth	January	FORM	NJ_haze_2002	4	1.38	1.12	-18.54	18.54	-20.50	20.50

Table 4-22. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for July at Camden Lab

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Camden	July	PAR	NJ_haze_2002	5	5.55	83.25	1400.61	1400.61	172.30	172.30
Camden	July	OLE	NJ_haze_2002	5	1.04	2.05	97.38	97.38	68.40	68.40
Camden	July	ALD2	NJ_haze_2002	5	0.66	2.92	340.45	340.45	128.40	128.40
Camden	July	TOL	NJ_haze_2002	5	1.53	2.07	35.41	108.33	44.20	83.40
Camden	July	XYL	NJ_haze_2002	5	0.62	1.11	78.62	78.62	53.30	53.30
Camden	July	FORM	NJ_haze_2002	5	2.04	4.38	114.47	114.47	80.00	80.00

Table 4-23. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for July at Rutgers University

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Rutgers	July	PAR	NJ_haze_2002	3	7.40	71.82	870.93	870.93	159.30	159.30
Rutgers	July	OLE	NJ_haze_2002	3	0.73	2.33	218.32	218.32	104.90	104.90
Rutgers	July	ALD2	NJ_haze_2002	3	2.09	3.20	53.04	54.22	39.10	40.20
Rutgers	July	TOL	NJ_haze_2002	3	1.07	1.55	44.79	48.38	36.80	40.70
Rutgers	July	XYL	NJ_haze_2002	3	1.10	0.87	-20.70	55.01	-11.10	65.50
Rutgers	July	FORM	NJ_haze_2002	3	6.17	4.86	-21.26	27.46	-24.50	31.30

Table 4-24. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for July at Chester

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Chester	July	PAR	NJ_haze_2002	2	6.43	57.61	796.65	796.65	155.10	155.10
Chester	July	OLE	NJ_haze_2002	2	0.52	1.89	267.38	267.38	112.90	112.90
Chester	July	ALD2	NJ_haze_2002	2	2.42	2.50	3.33	21.59	0.00	21.20
Chester	July	TOL	NJ_haze_2002	2	1.63	0.94	-42.46	67.02	-38.20	77.30
Chester	July	XYL	NJ_haze_2002	2	1.75	0.51	-70.94	70.94	-90.90	90.90
Chester	July	FORM	NJ_haze_2002	2	9.36	4.23	-54.80	54.80	-76.10	76.10

Table 4-25. Summary of regional haze model performance evaluation results against 2002 NJ Air Toxics data for July at Elizabeth

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Elizabeth	July	PAR	NJ_haze_2002	6	9.55	100.57	953.27	953.27	164.20	164.20
Elizabeth	July	OLE	NJ_haze_2002	6	4.55	2.35	-48.27	66.28	-13.70	66.50
Elizabeth	July	ALD2	NJ_haze_2002	6	1.64	3.28	100.19	100.19	66.10	66.10
Elizabeth	July	TOL	NJ_haze_2002	6	1.32	3.36	154.31	154.31	83.90	83.90
Elizabeth	July	XYL	NJ_haze_2002	6	0.88	2.36	169.50	169.50	92.70	92.70
Elizabeth	July	FORM	NJ_haze_2002	6	4.12	4.33	5.08	16.82	1.60	16.60

Box plots

Box plots showing average monthly concentrations at NJ Air Toxics network sites and average predicted monthly concentrations are presented in Figure 4-2 through Figure 4-7. These charts illustrate that the model is able to reproduce the general pattern of seasonal concentration change, although the levels of the species are often overpredicted

by a factor of two or more. The box plots show first and third quartile values as the bottom and top of the boxes, respectively, and the central line represents the median value for both observed and predicted concentrations.

The model is fairly accurate for lower, winter concentrations of acetaldehyde (and other higher aldehydes) as shown in Figure 4-2, but higher, summer concentrations tailing into the fall are overestimated. Though the month-to-month trends for formaldehyde are represented well in the model, the model has some difficulty in reproducing the observed values seen in the late winter and early spring, as shown in Figure 4-3. Monthly patterns for both olefins and toluene are well reproduced as well, though consistently overestimated by a factor of two or more, as seen in Figure 4-4 and Figure 4-6. Summer concentrations of xylenes are adequately represented (though still overpredicted), but winter concentrations are grossly overestimated (Figure 4-7), as are those for paraffins year-round (Figure 4-5).

Average diurnal trends (July only) from the PAMS network are compared against model predictions in box plots in Figure 4-8 through Figure 4-14. Similar to the monthly box plots, these plots show that while observed CB4 species patterns are generally well reproduced in the model, the levels are often grossly overpredicted. Overpredictions seem to be worst during morning peak hours, and are persistent over the nighttime hours, indicating that formation and destruction rate chemistry and/or advection are not well represented in the model. Predicted concentrations of higher aldehydes are much higher (about one order of magnitude) than observations indicate (Figure 4-8). Ethylene is overpredicted by a factor of about two, as shown in Figure 4-9, though the diurnal pattern is fairly accurate. Isoprene, which is primarily emitted from trees and plants, is well predicted at night (close to 0 ppb), but highly overpredicted in the daytime (Figure 4-10). Such overprediction indicates that either emission levels are too high or modeled vertical/horizontal transport is insufficient to dilute the isoprene (and the other species) to observed levels. Modeled olefin levels show the converse trend, with low midday values and high nighttime values, though this pattern is largely unobserved (Figure 4-11). The diurnal pattern for paraffins, toluene, and xylenes are well reproduced by the model, as shown in Figure 4-12, Figure 4-13, and Figure 4-14, respectively, though the concentrations are from two to four times too high. These compounds show similar diurnal trends, with higher and accumulating nighttime concentrations followed by daytime lows.

Figure 4-2. Comparison of monthly average higher aldehyde concentrations for the NJ Air Toxics network at all sites for the regional haze model

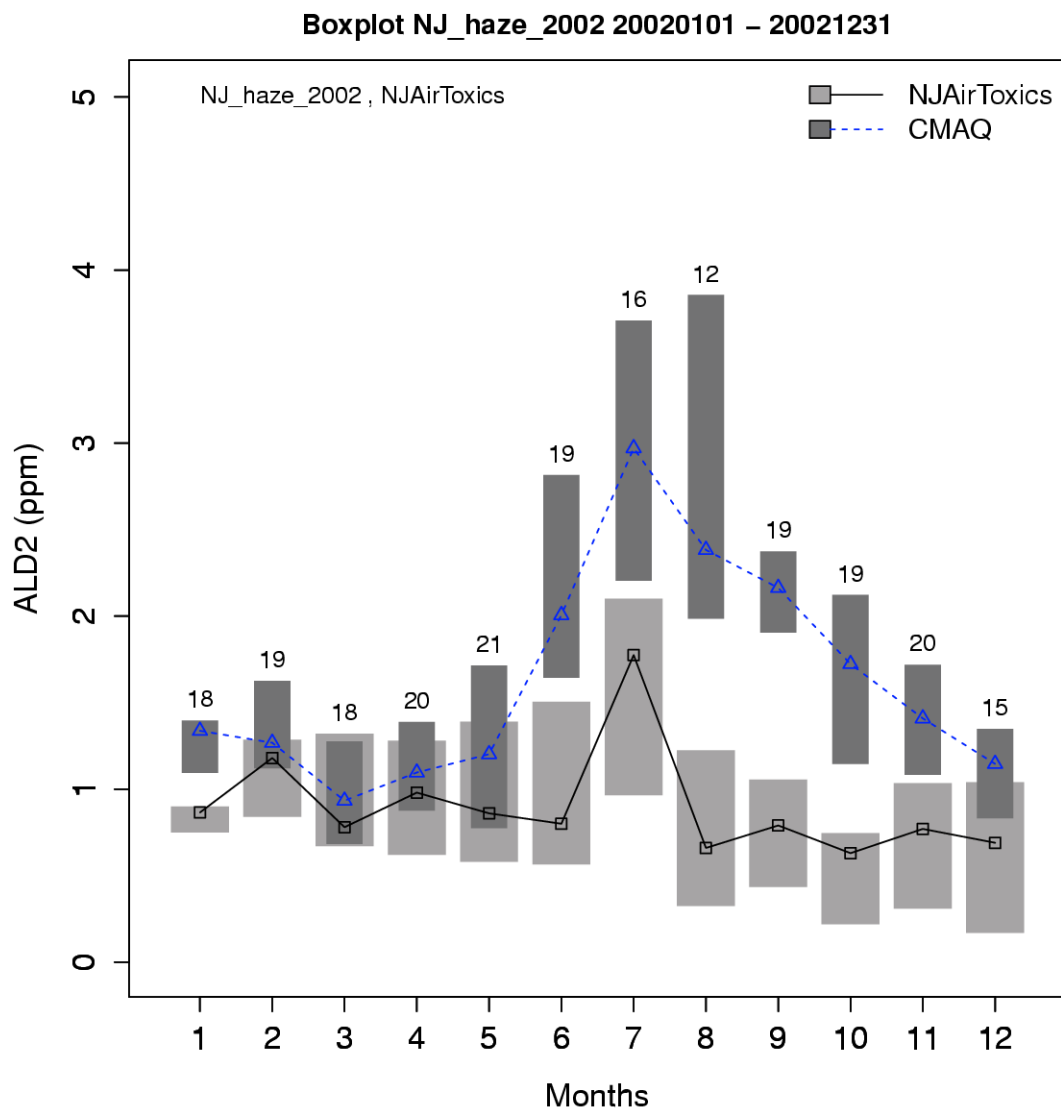


Figure 4-3. Comparison of monthly average formaldehyde concentrations for the NJ Air Toxics network at all sites for the regional haze model

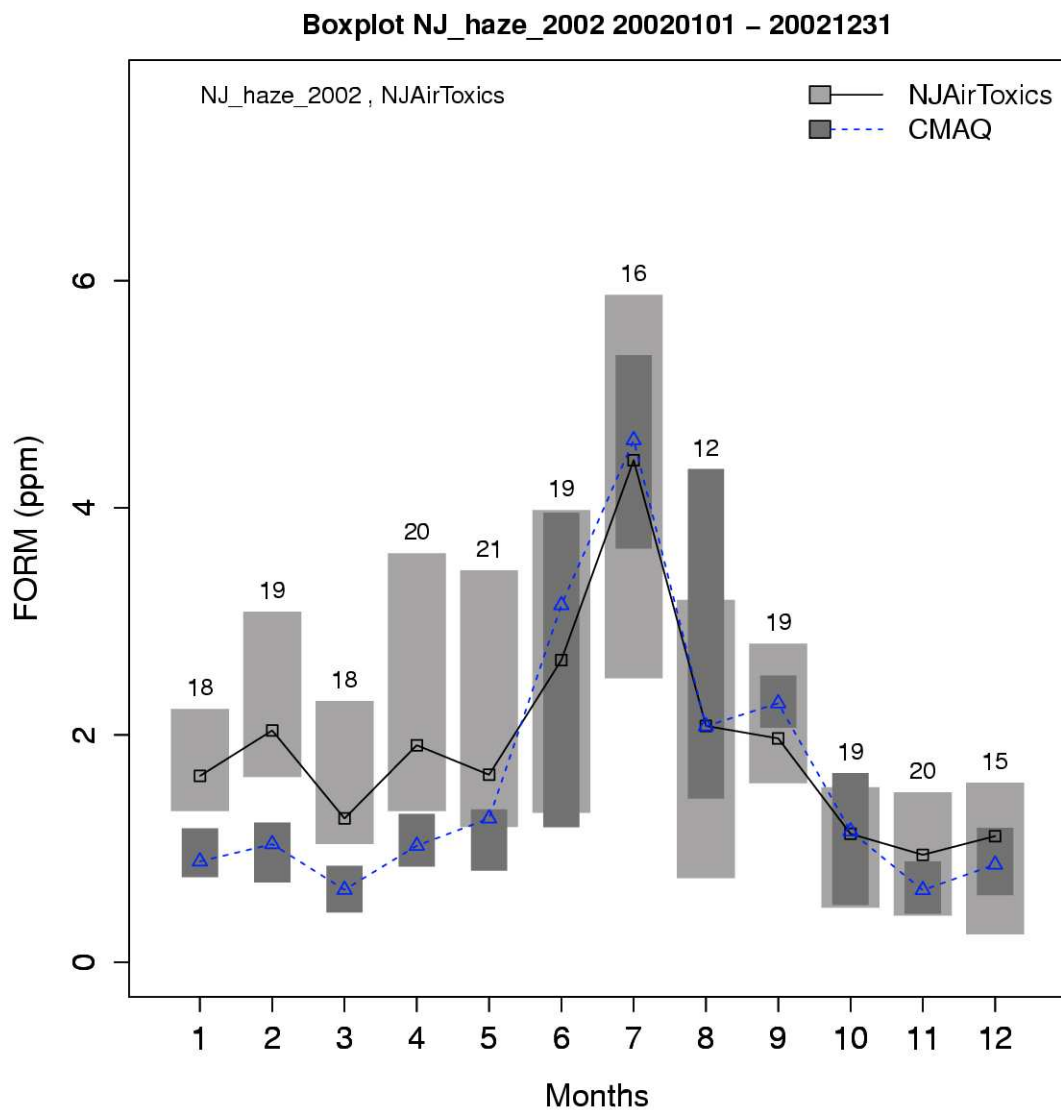


Figure 4-4. Comparison of monthly average olefin concentrations for the NJ Air Toxics network at all sites for the regional haze model

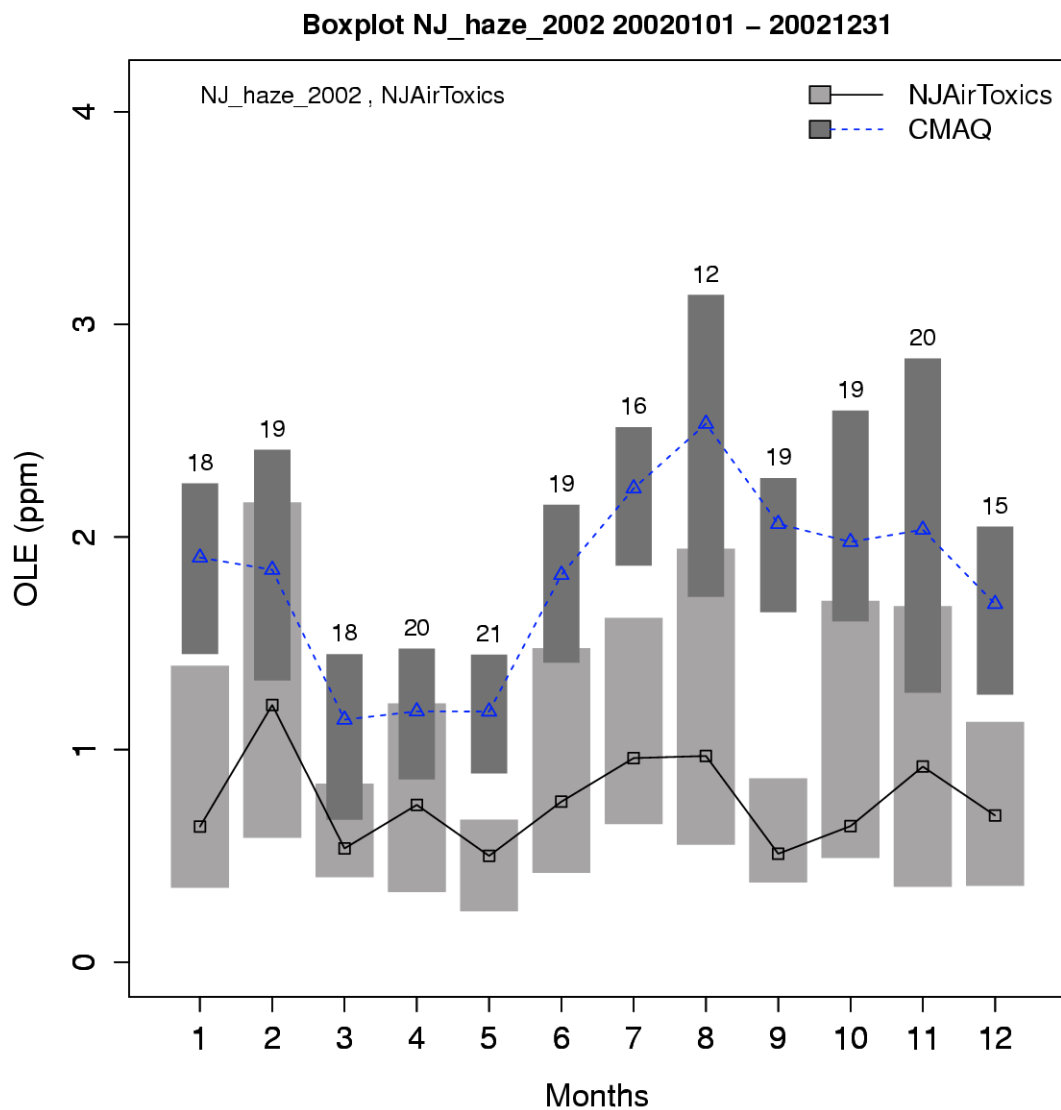


Figure 4-5. Comparison of monthly average paraffin concentrations for the NJ Air Toxics network at all sites for the regional haze model

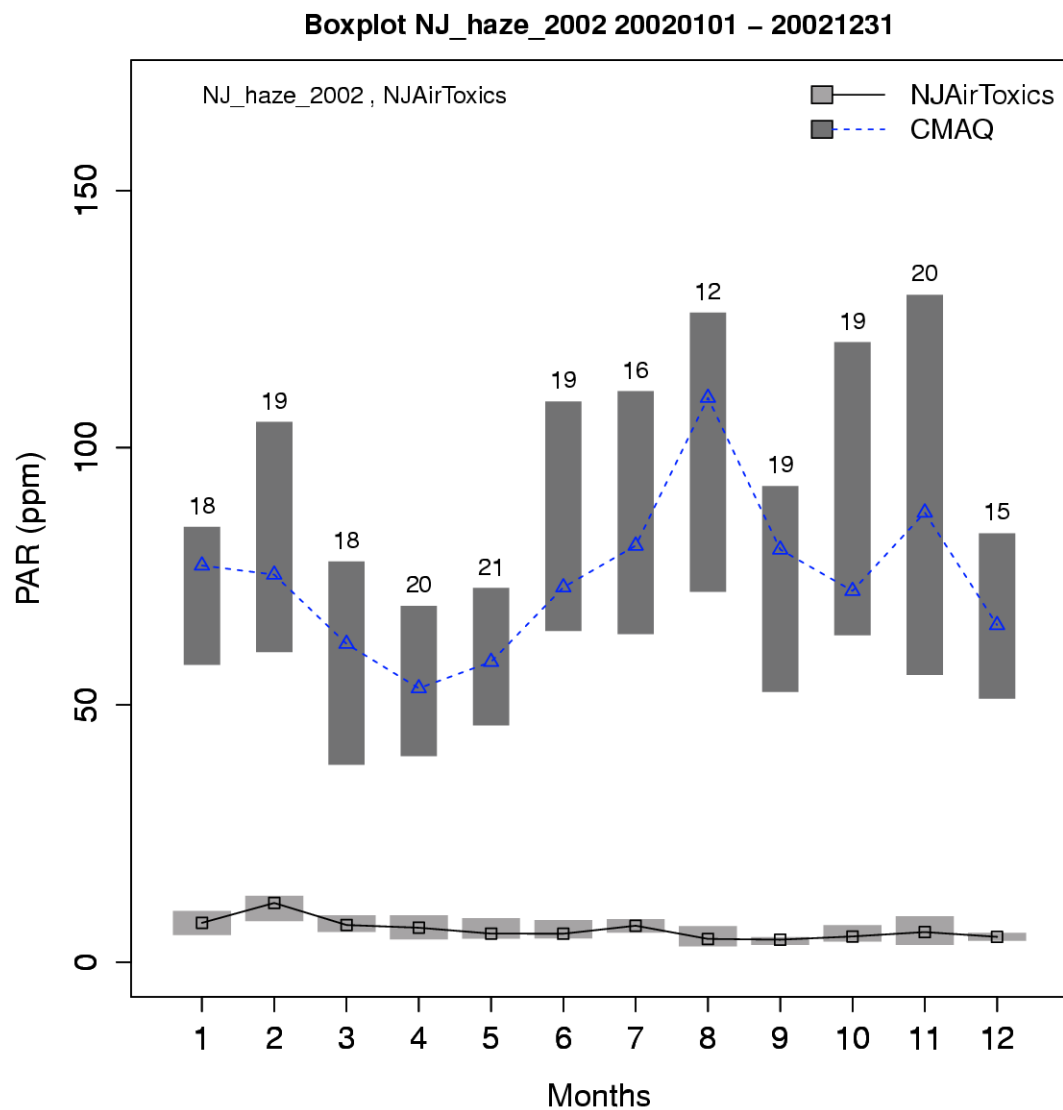


Figure 4-6. Comparison of monthly average toluene concentrations for the NJ Air Toxics network at all sites for the regional haze model

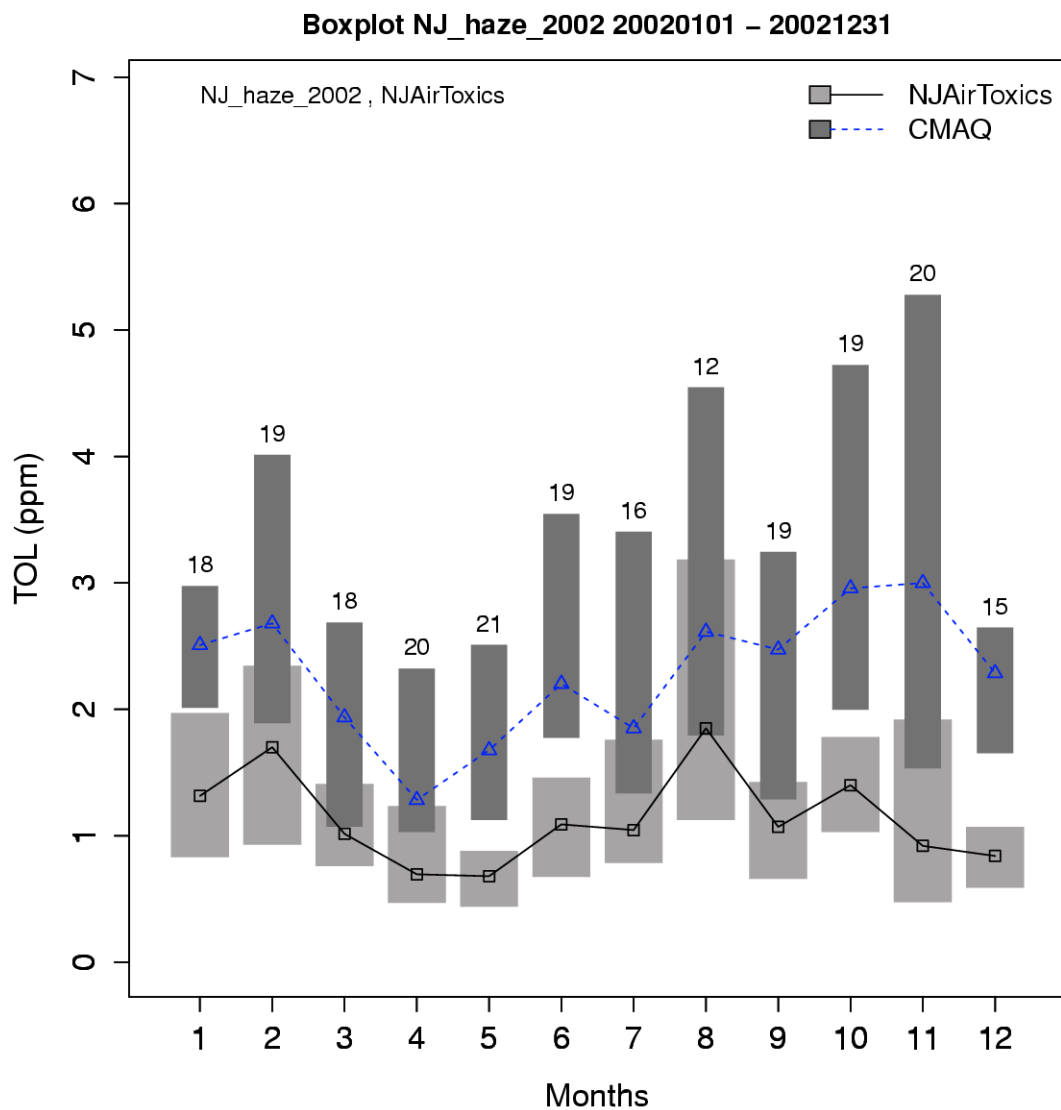


Figure 4-7. Comparison of monthly average xylenes concentrations for the NJ Air Toxics network at all sites for the regional haze model

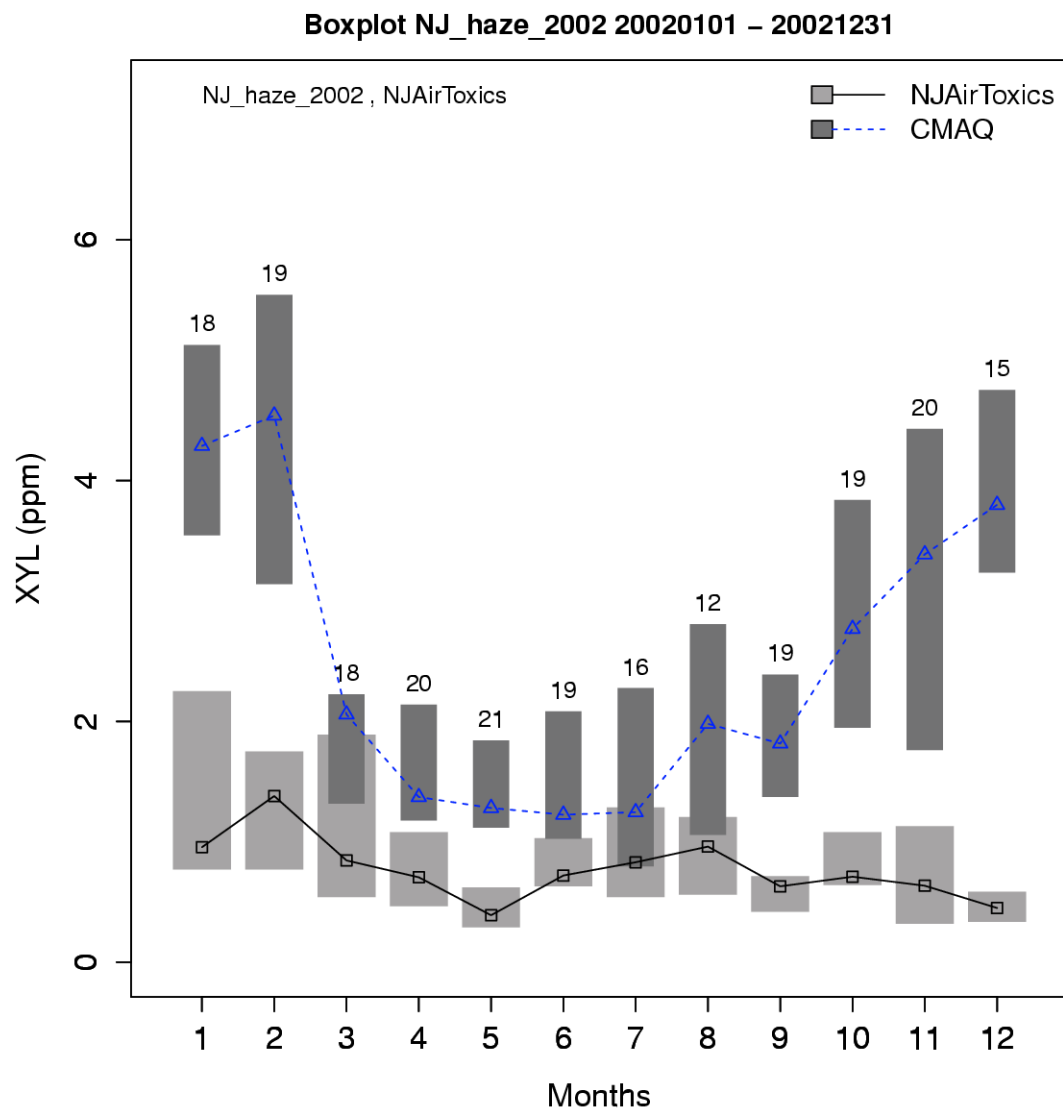


Figure 4-8. Comparison of July diurnal patterns for higher aldehydes from the PAMS network at all sites for the regional haze model

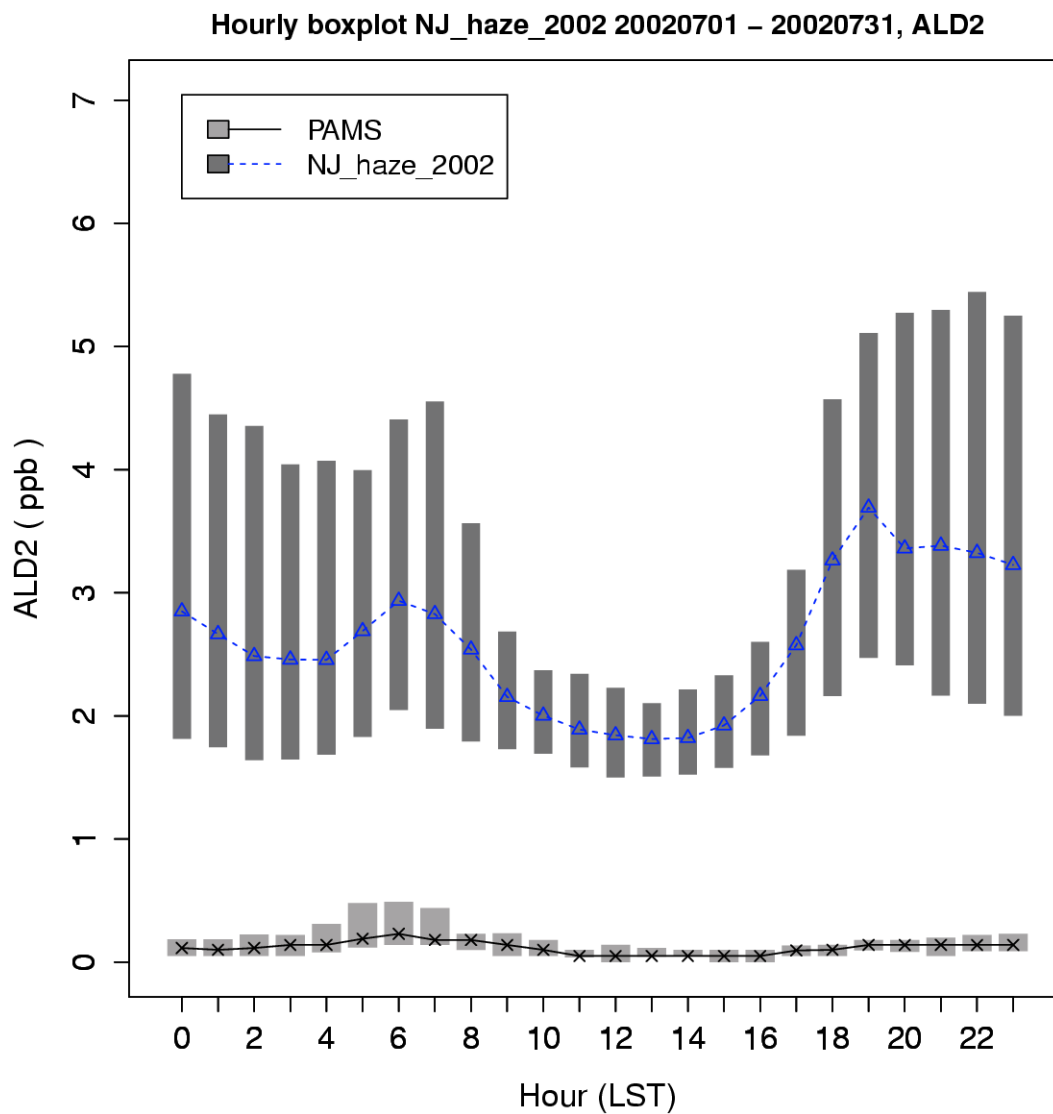


Figure 4-9. Comparison of July diurnal patterns for ethylene from the PAMS network at all sites for the regional haze model

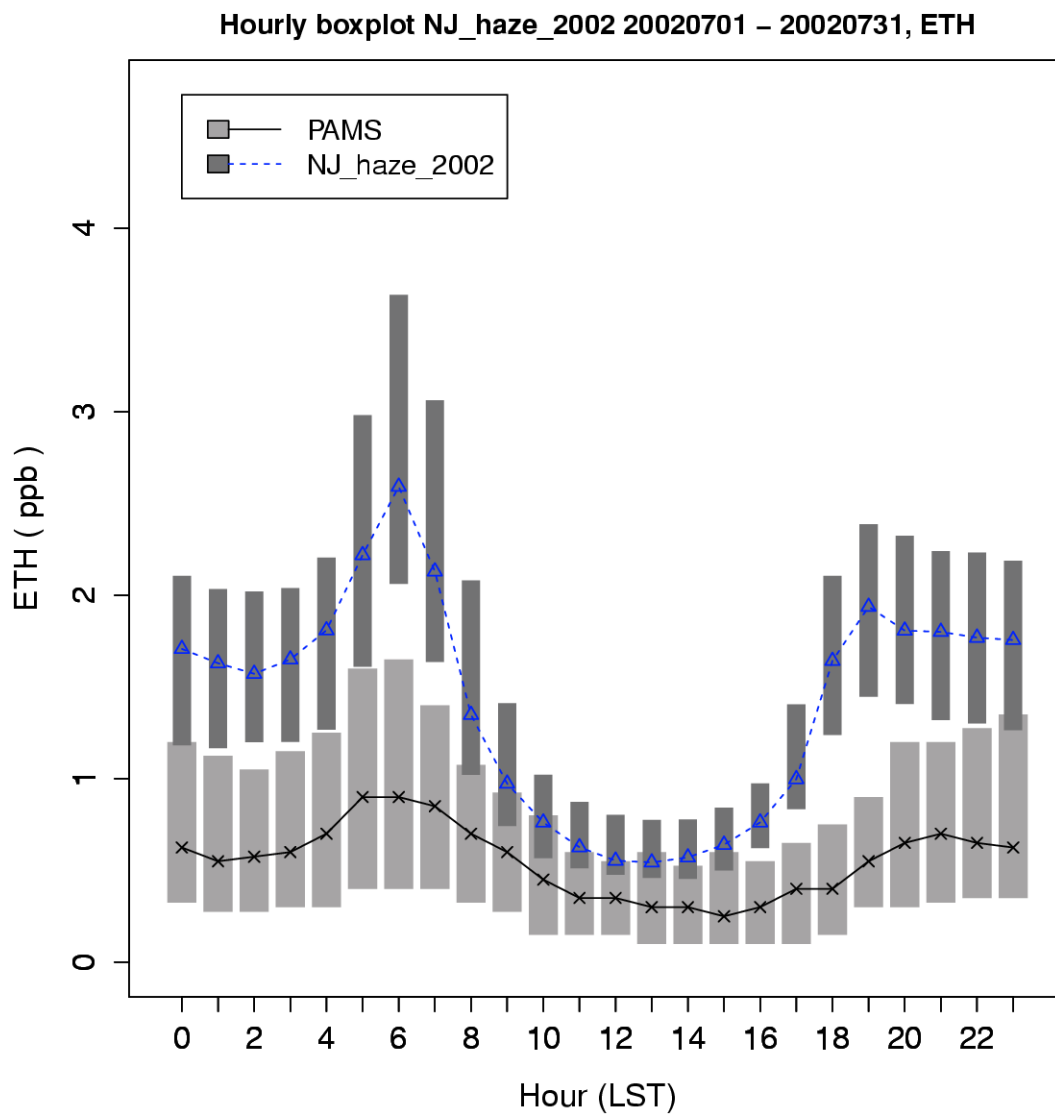


Figure 4-10. Comparison of July diurnal patterns for isoprene from the PAMS network at all sites for the regional haze model

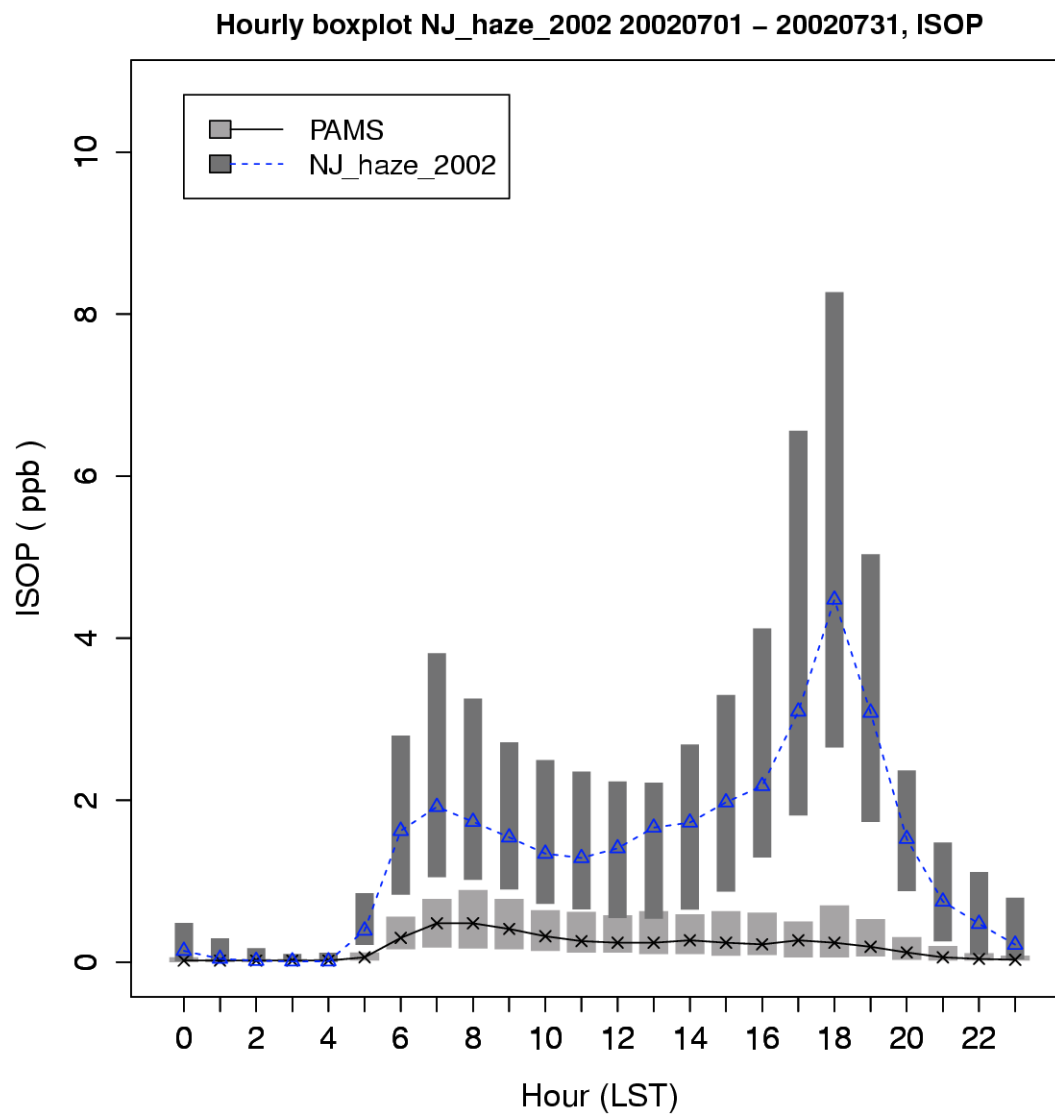


Figure 4-11. Comparison of July diurnal patterns for olefins from the PAMS network at all sites for the regional haze model

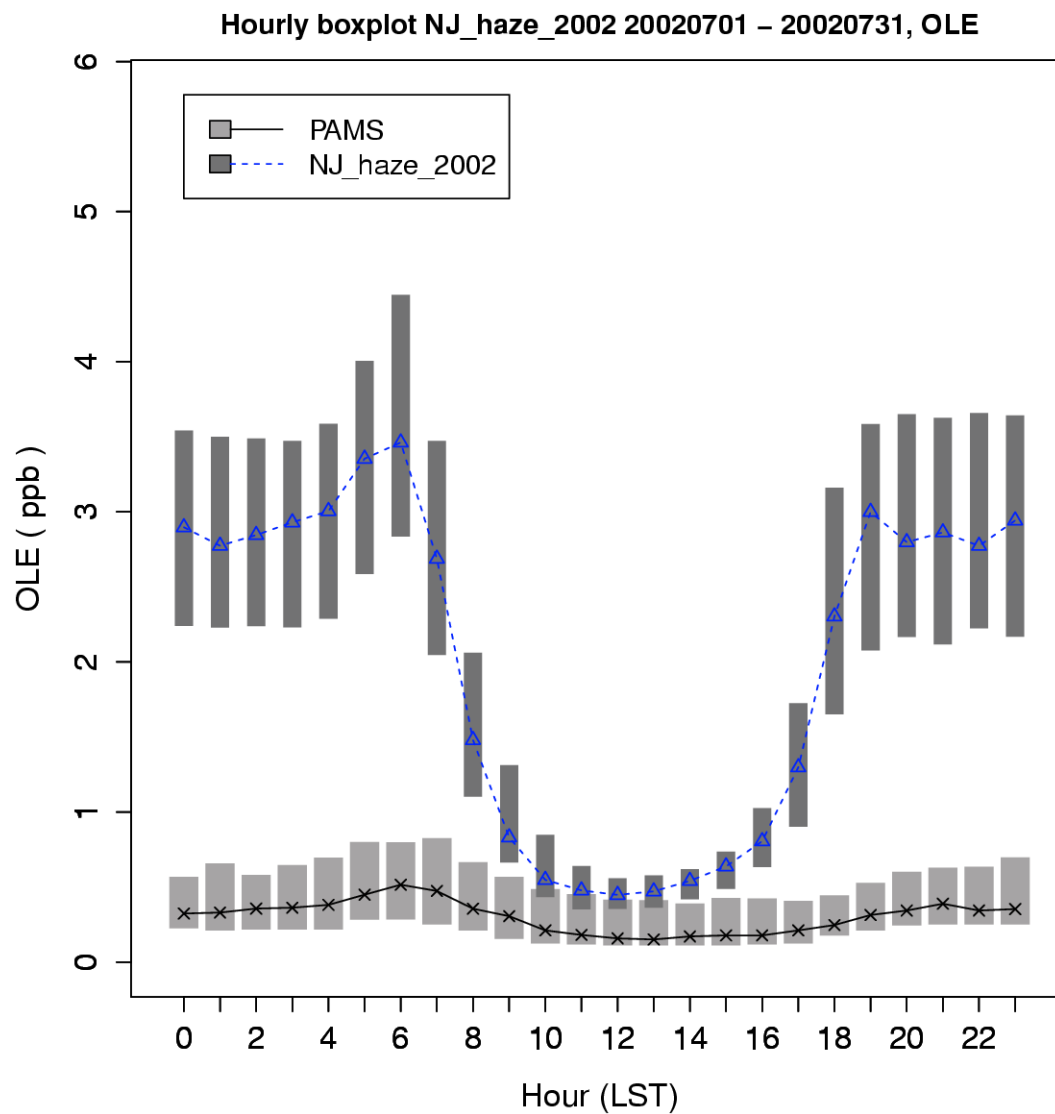


Figure 4-12. Comparison of July diurnal patterns for paraffins from the PAMS network at all sites for the regional haze model

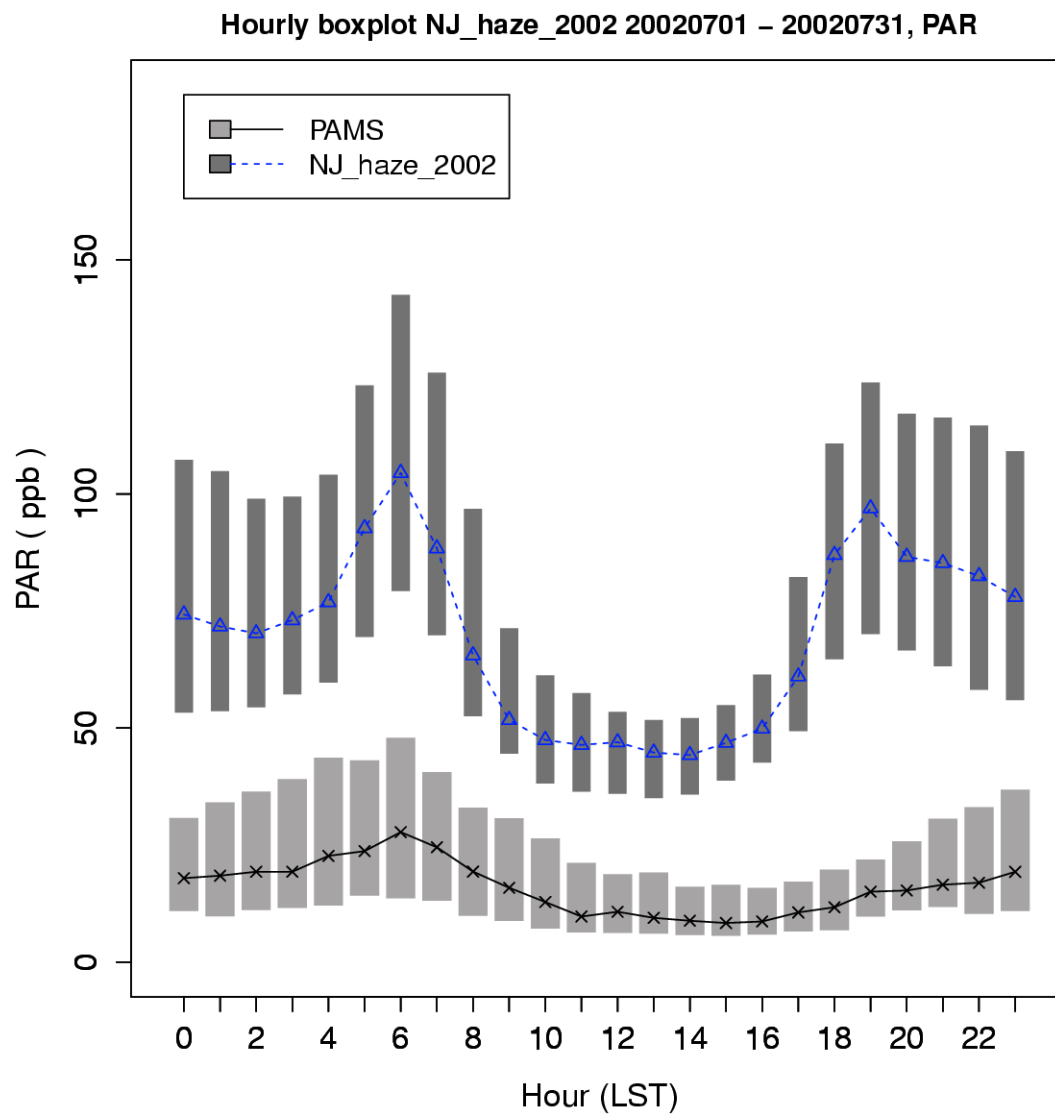


Figure 4-13. Comparison of July diurnal patterns for toluene from the PAMS network at all sites for the regional haze model

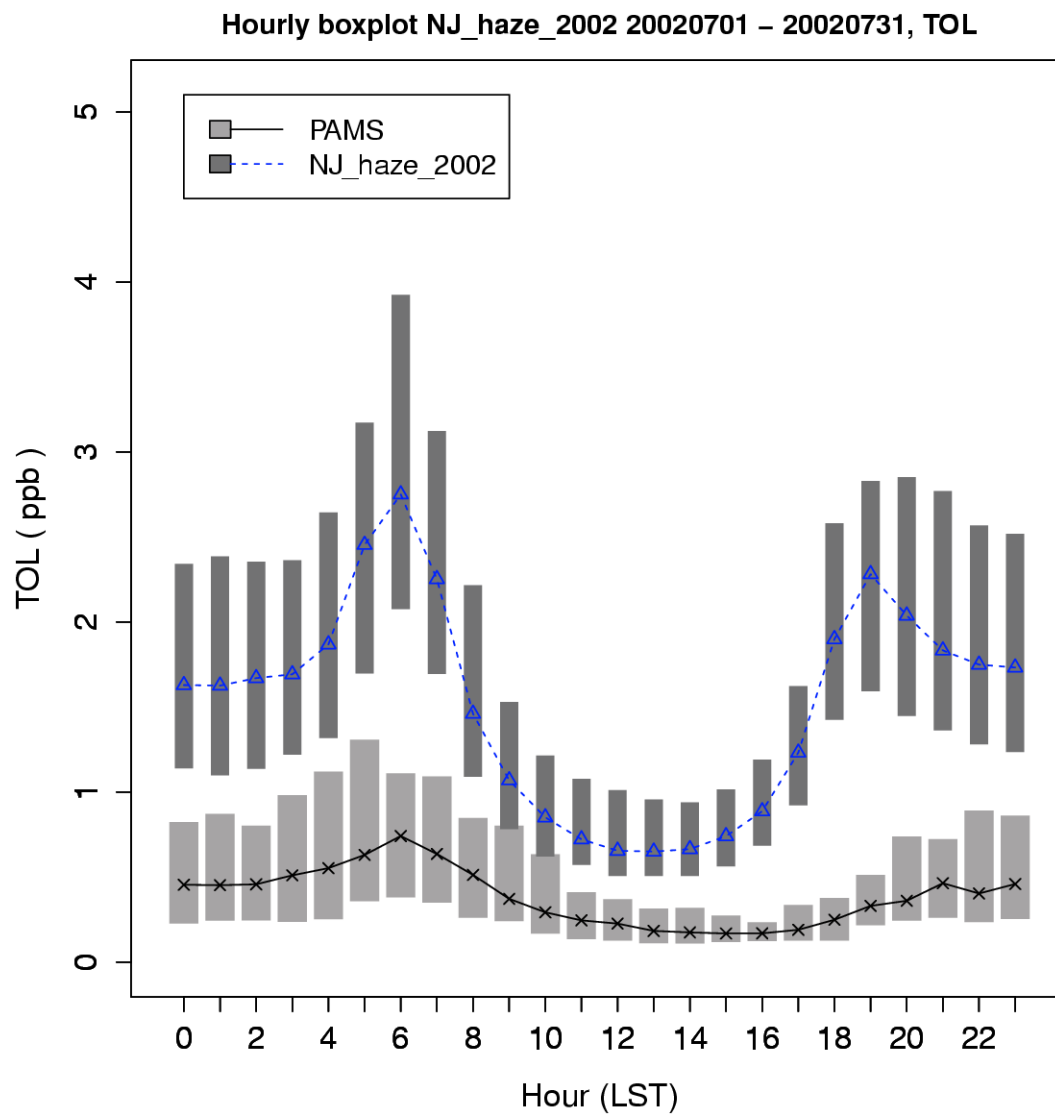
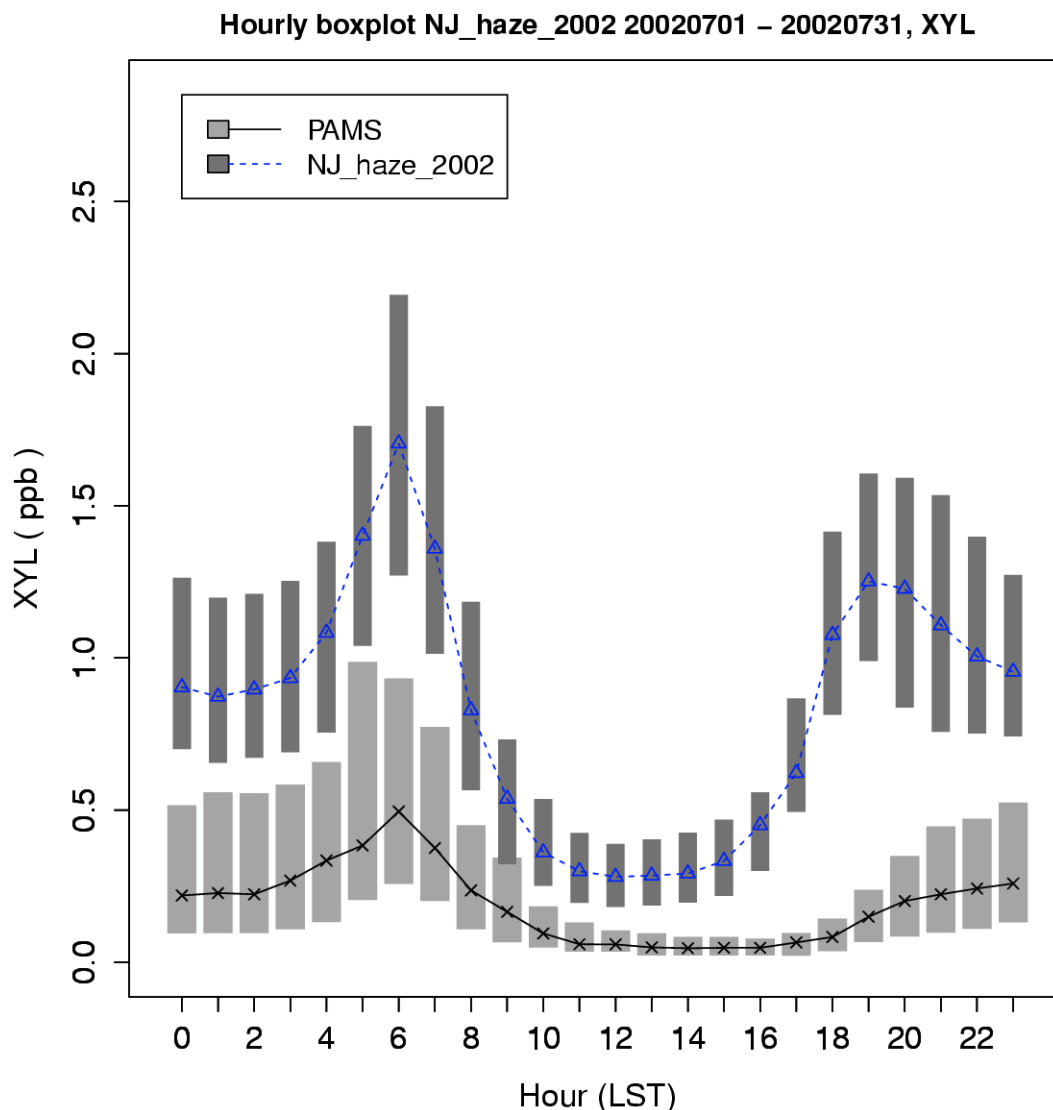


Figure 4-14. Comparison of July diurnal patterns for xylenes from the PAMS network at all sites for the regional haze model



Scatter plots

Results are shown for January and July only for the NJ Air Toxics network. Data are paired in space and time. Data are plotted with the observational value on the x-axis and modeled data on the y-axis. Perfect agreement between the model and observations would be seen as a series of points along the 45-degree line.

Scatter plots for the winter generally show poor agreement for the model against observed concentrations, as shown in Figure 4-15 through Figure 4-20. Aldehydes (shown in Figure 4-15) appear to be better predicted at higher concentrations; lower observed concentrations are not well-predicted by the model. Conversely, for formaldehyde (see Figure 4-16), lower concentrations are better predicted than higher observed concentrations. Olefins are generally well predicted (though somewhat

overpredicted at lower concentration levels), but the model failed to predict the highest observed concentration, which greatly affected bias and error metrics (see Figure 4-17). Paraffins are almost universally overpredicted; observed levels rarely exceed 20 ppb while modeled levels are often in excess of 70 ppb (Figure 4-18). Similarly, xylenes are also almost universally overpredicted (Figure 4-20). Toluene (Figure 4-19) is more like formaldehyde, with lower concentrations achieving better predictions than higher concentrations, which appear to be significantly underestimated. Such large underestimates may be the result of a gap in emission inventories.

Summertime scatter plots paired in space and time (Figure 4-21 through Figure 4-26) show different patterns of agreement between observations and predictions than the winter scatter plots do for some species, and similar patterns for others. As with winter levels, for higher aldehydes (Figure 4-21), the model shows better agreement at higher concentration levels than at lower levels. Unlike in the winter, formaldehyde (Figure 4-22) is well represented in the model, which shows no consistent bias at higher or lower values. Summertime olefin values (Figure 4-23) are well represented in the model at lower concentration levels, and poorly reproduced at higher observed levels. Summer levels of paraffins are even more grossly overpredicted (Figure 4-24) than in the winter. Levels of toluene (Figure 4-25) and xylenes (Figure 4-26) are often overpredicted, though there appears to be no consistent trend for which values (high or low) suffer worst from this effect.

Figure 4-15. Comparison of January scatter plot for higher aldehydes from NJ Air Toxics sites for the regional haze model

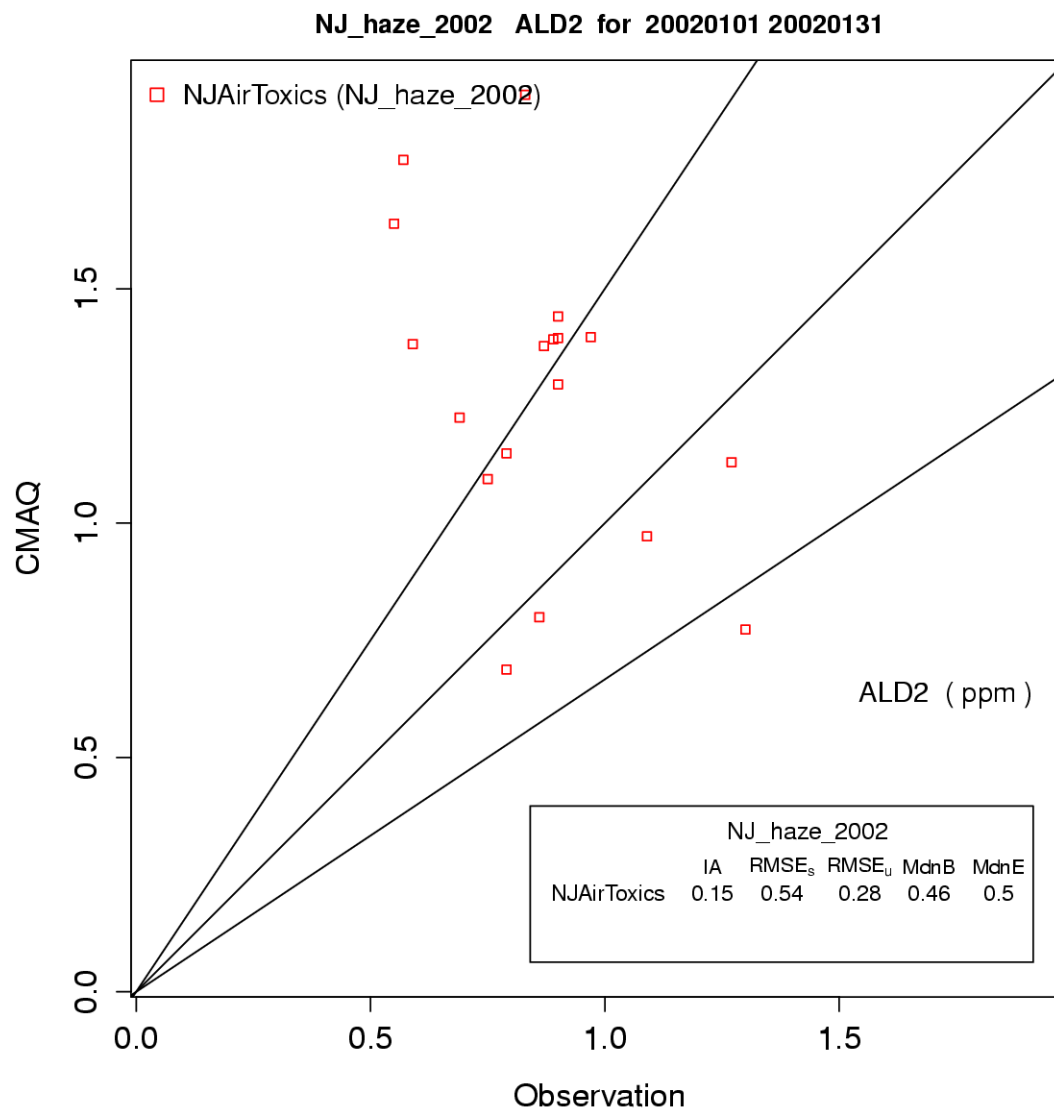


Figure 4-16. Comparison of January scatter plot for formaldehyde from NJ Air Toxics sites for the regional haze model

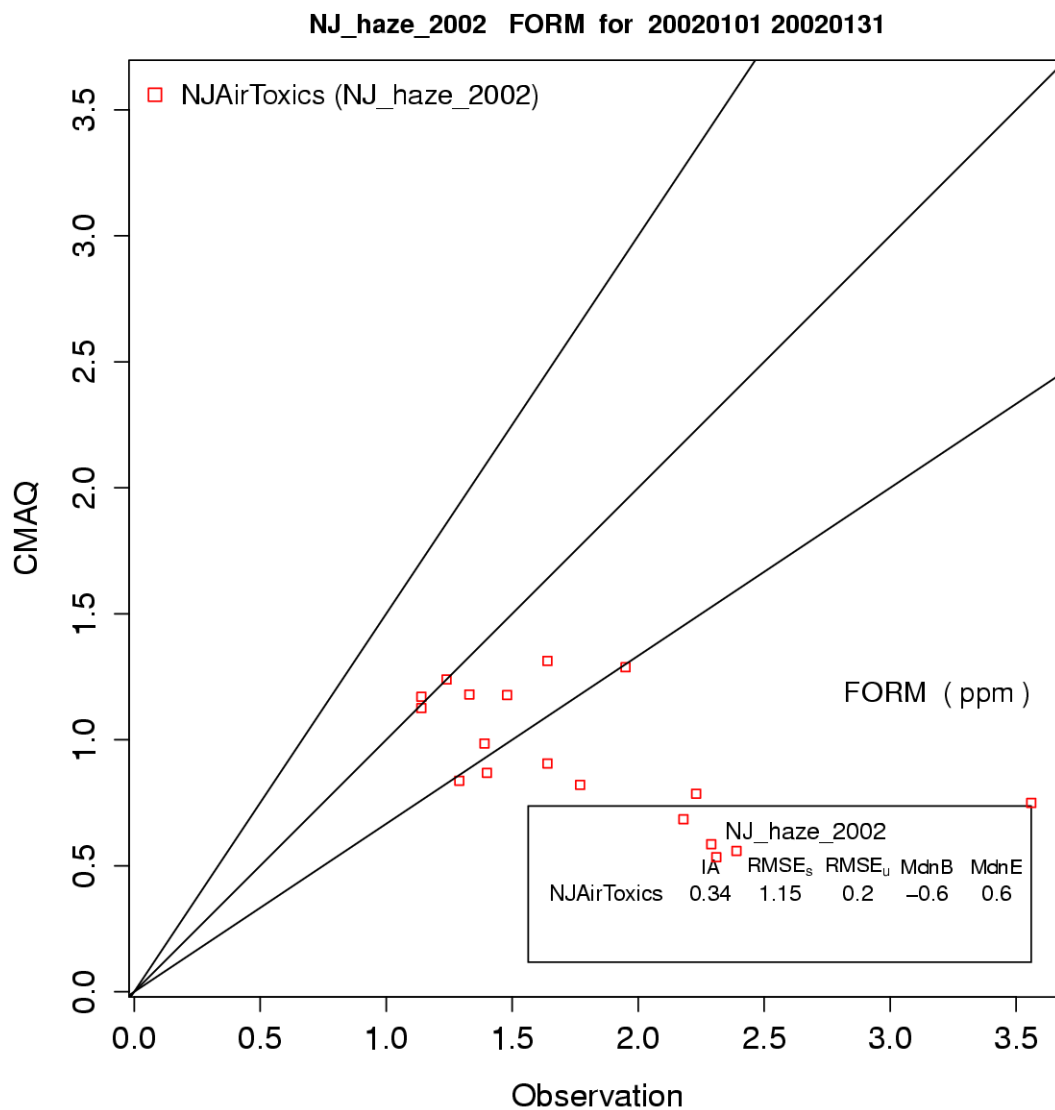


Figure 4-17. Comparison of January scatter plot for olefins from NJ Air Toxics sites for the regional haze model

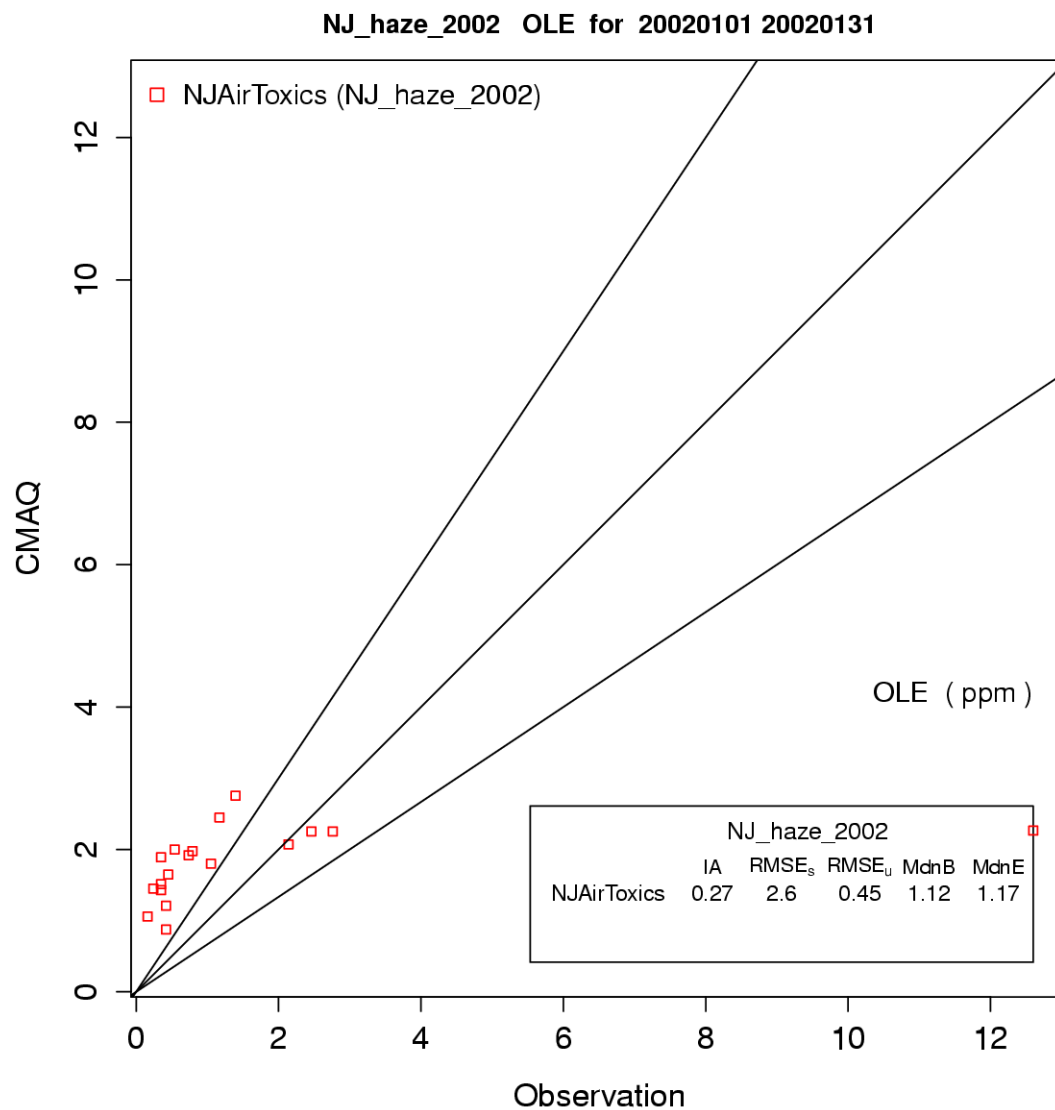


Figure 4-18. Comparison of January scatter plot for paraffins from NJ Air Toxics sites for the regional haze model

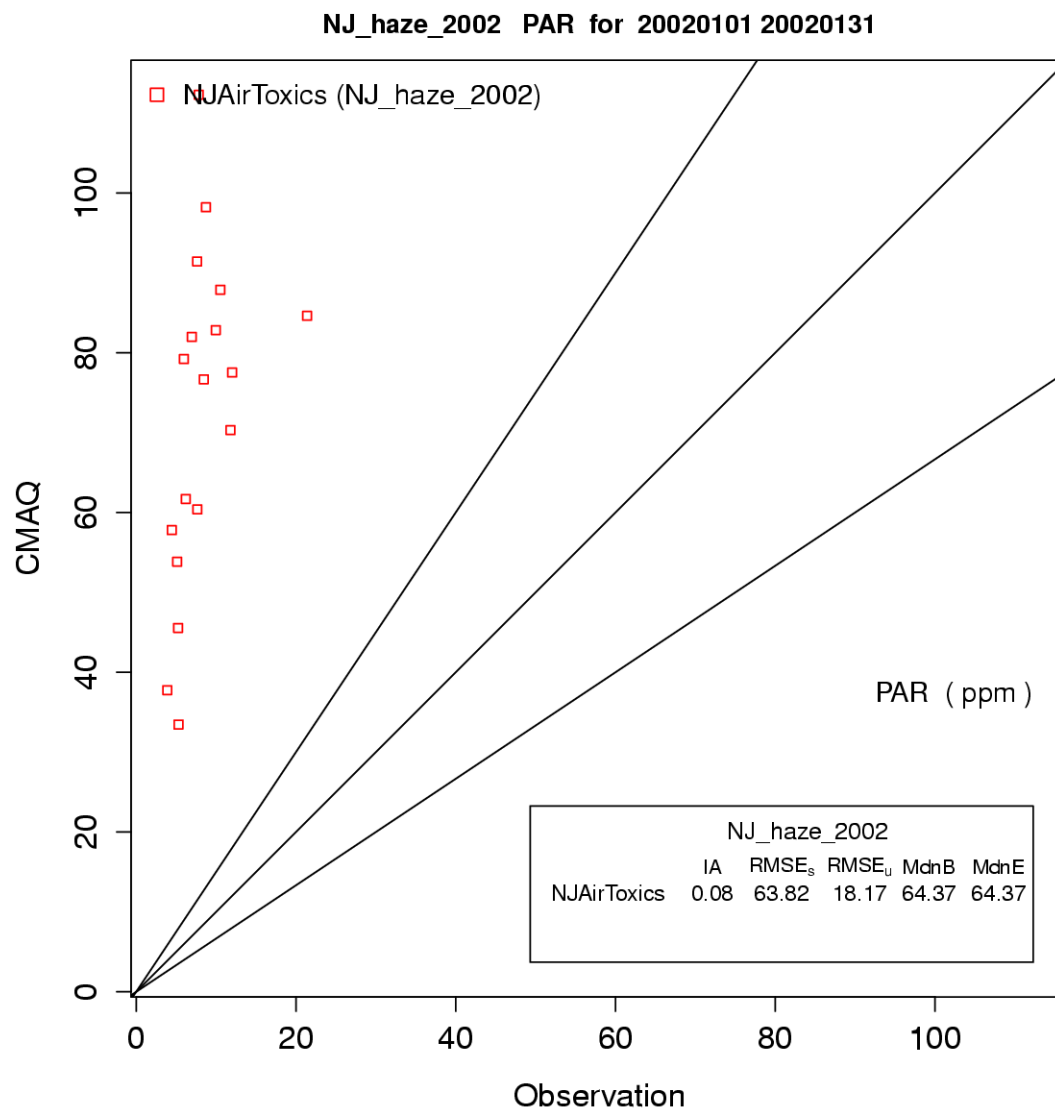


Figure 4-19. Comparison of January scatter plot for toluene from NJ Air Toxics sites for the regional haze model

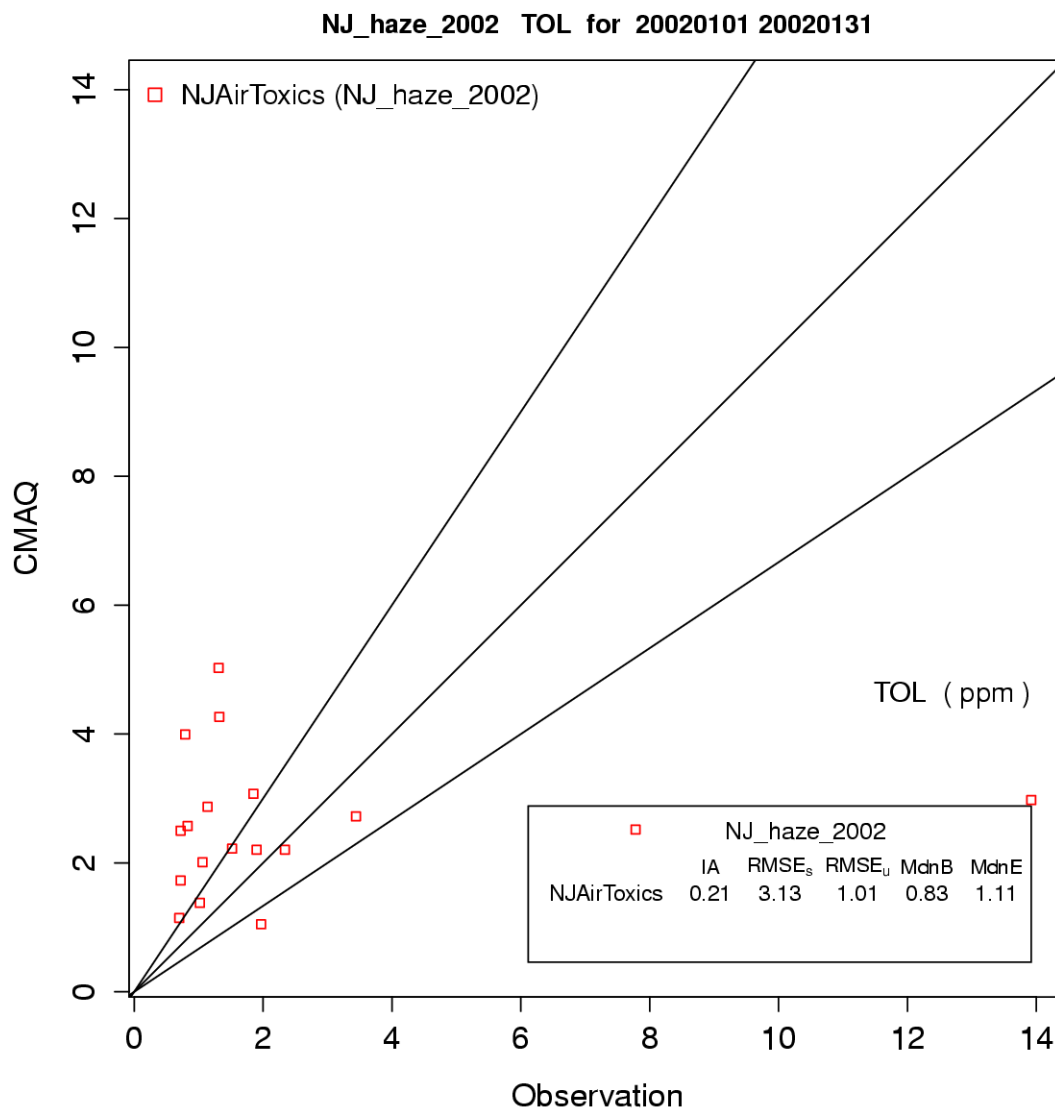


Figure 4-20. Comparison of January scatter plot for xylenes from NJ Air Toxics sites for the regional haze model

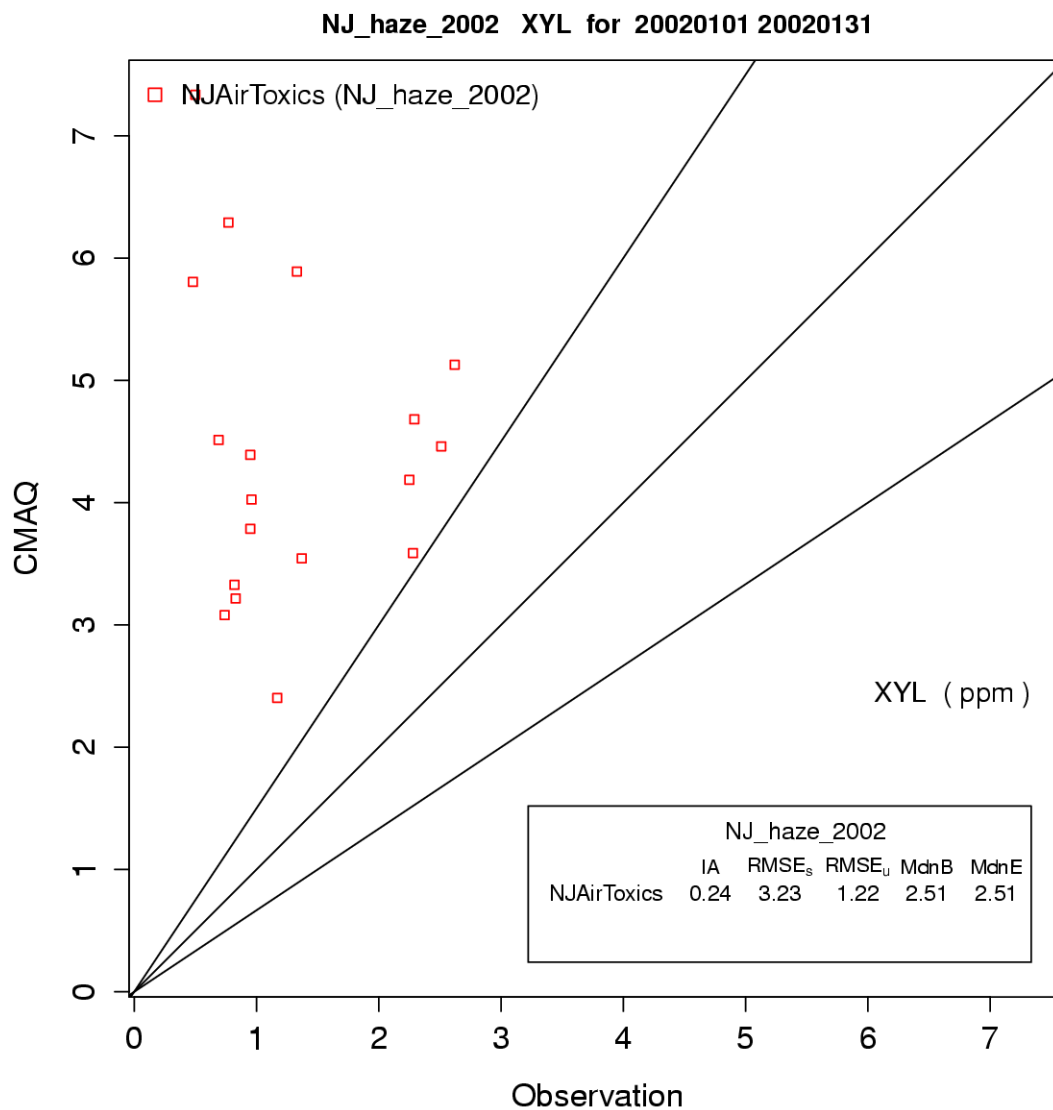


Figure 4-21. Comparison of July scatter plot for higher aldehydes from NJ Air Toxics sites for the regional haze model

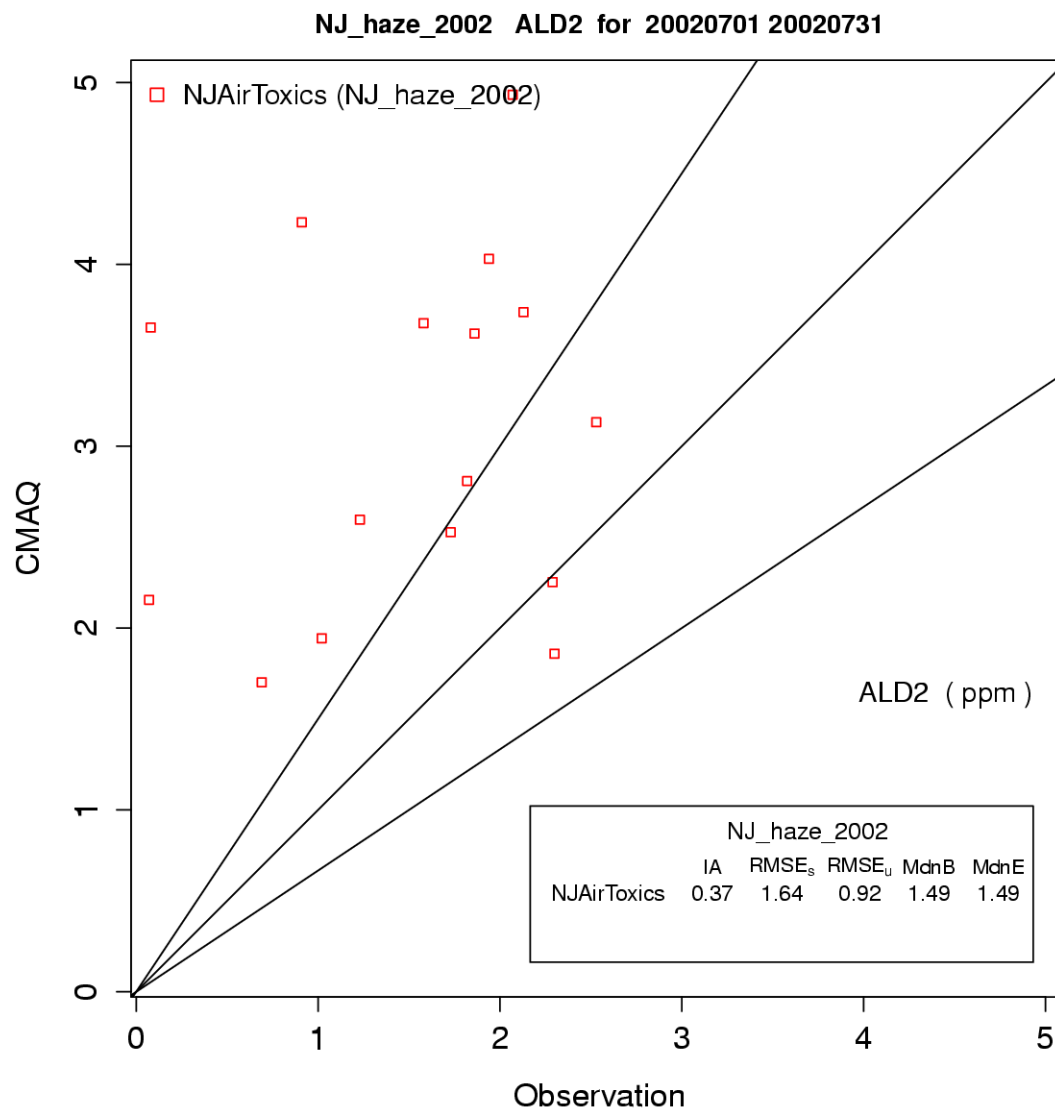


Figure 4-22. Comparison of July scatter plot for formaldehyde from NJ Air Toxics sites for the regional haze model

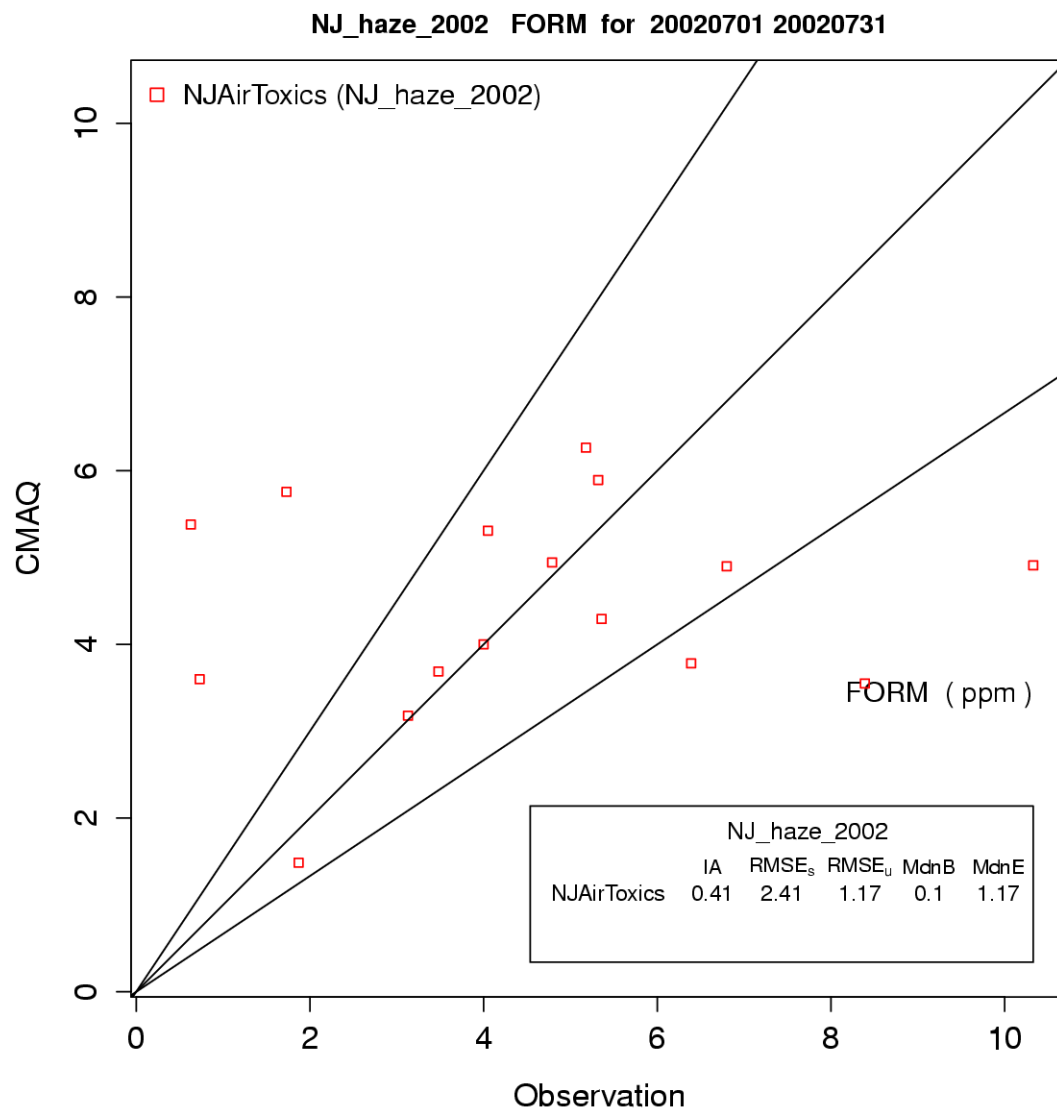


Figure 4-23. Comparison of July scatter plot for olefins from NJ Air Toxics sites for the regional haze model

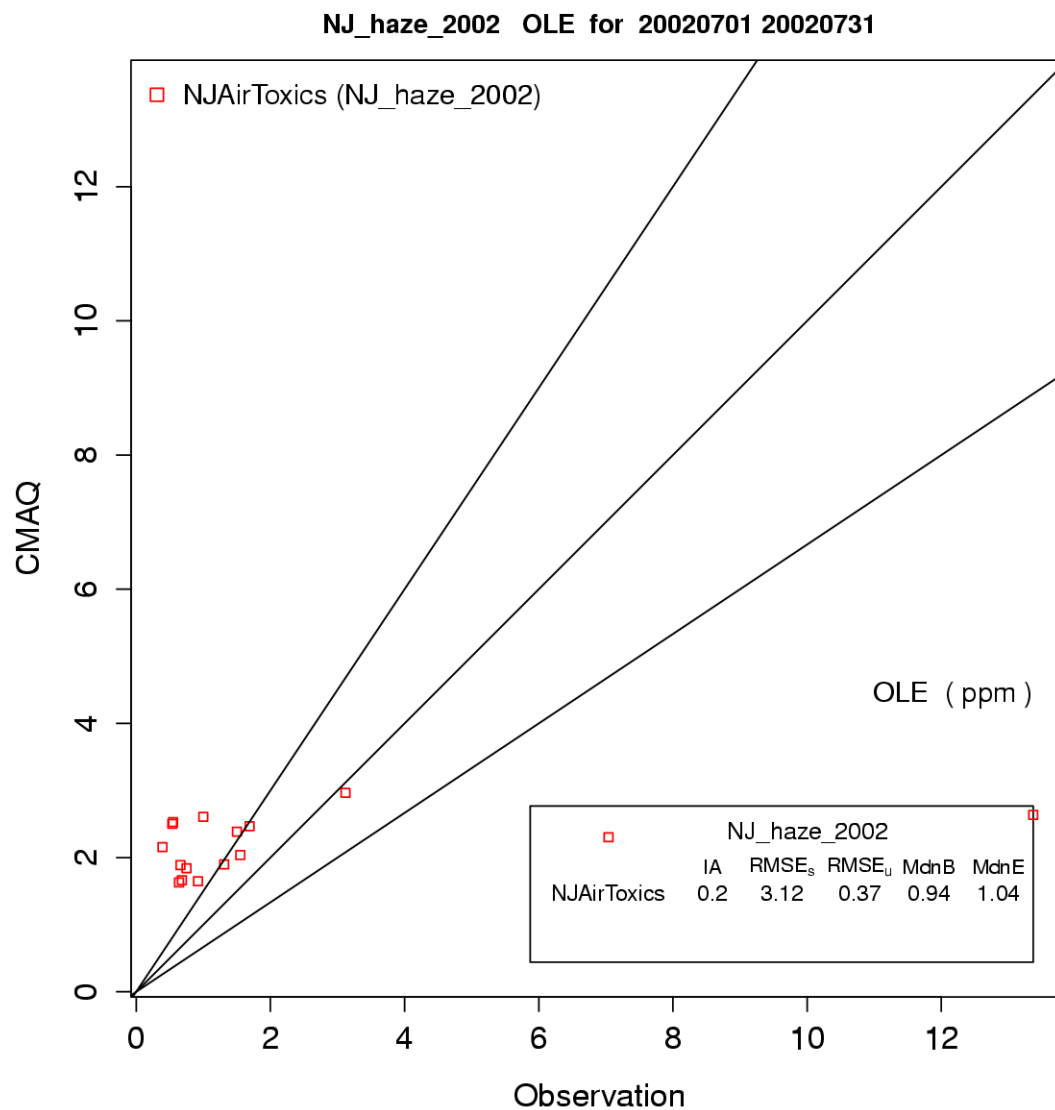


Figure 4-24. Comparison of July scatter plot for paraffins from NJ Air Toxics sites for the regional haze model

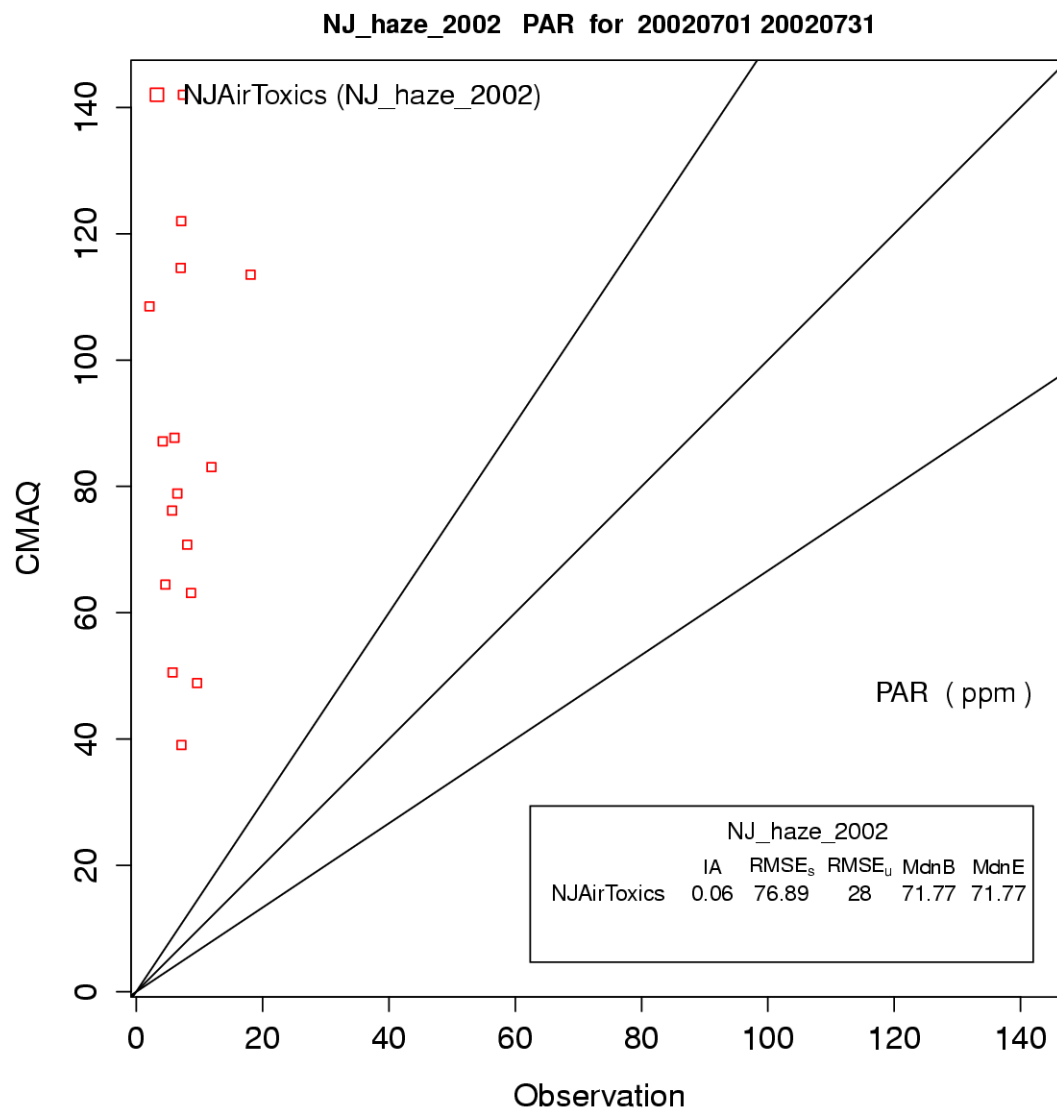


Figure 4-25. Comparison of July scatter plot for toluene from NJ Air Toxics sites for the regional haze model

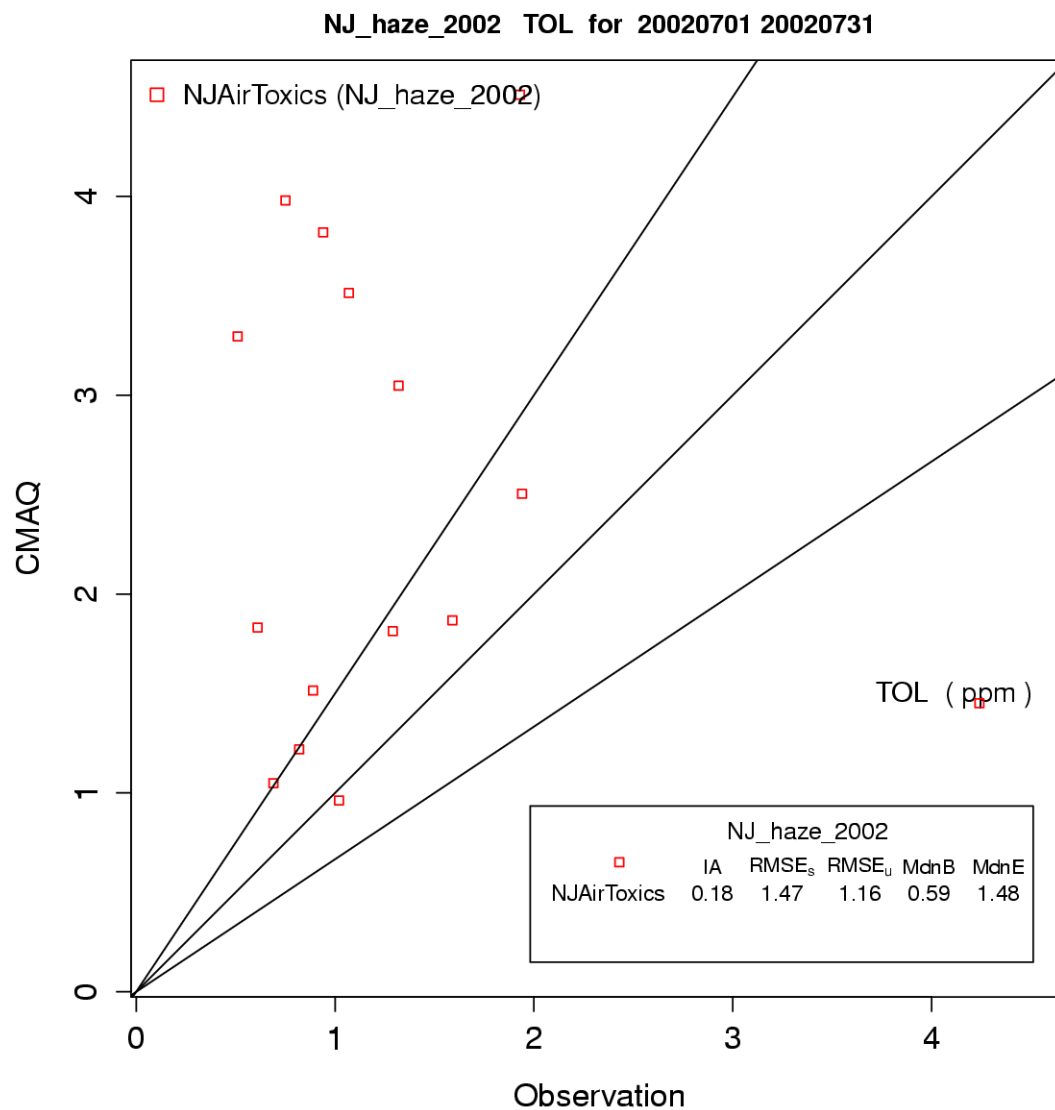
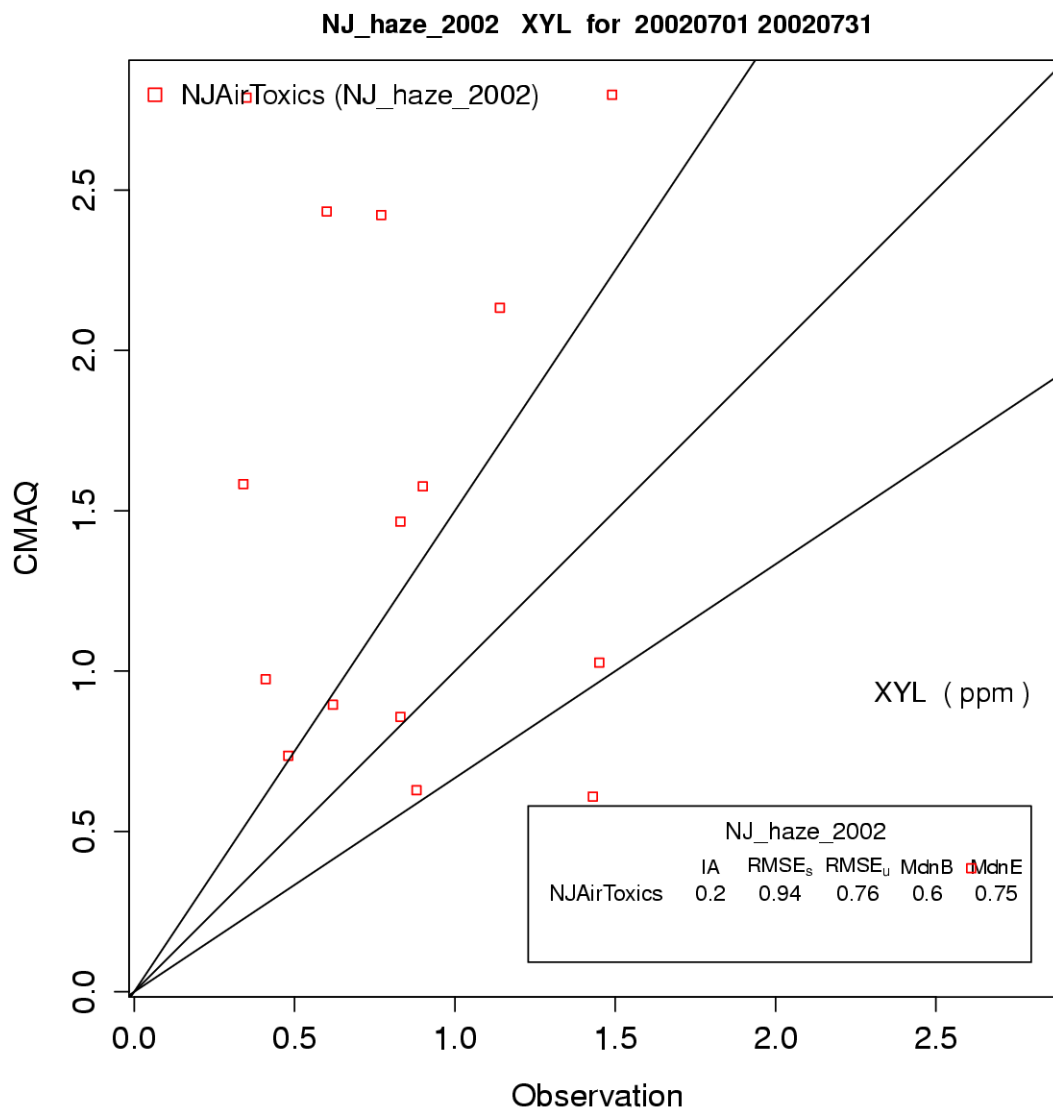


Figure 4-26. Comparison of July scatter plot for xylenes from NJ Air Toxics sites for the regional haze model



4.5.2. Air toxics model evaluation results

This section presents results of the model evaluation from the air toxics model (labeled NJ_tox_2002). Results are presented in the form of summary statistics for domain-wide (all monitors) and specific monitors, as well as in box plots, which help characterize the model versus observed temporal patterns, and scatter plots that show accuracy and precision of the model predictions.

Statistics

Domain-wide results

Table 4-26. Domain-wide summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for January

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
January	PAR	NJ_tox_2002	18	8.284	96.384	1063.44	1063.44	167.8	167.8
January	OLE	NJ_tox_2002	18	1.577	2.61	65.56	130.9	94.9	107.9
January	ALD2	NJ_tox_2002	18	0.862	1.67	93.76	93.76	61.3	61.3
January	TOL	NJ_tox_2002	18	2.463	3.069	24.6	105.89	51	85.7
January	XYL	NJ_tox_2002	18	1.306	1.935	48.15	83.4	38	63.9
January	FORM	NJ_tox_2002	18	1.798	1.399	-22.19	39.34	-22.9	42.7

Table 4-27. Domain-wide summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for July

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
July	PAR	NJ_tox_2002	13	8.032	92.925	1057.01	1057.01	165.7	165.7
July	OLE	NJ_tox_2002	13	2.388	1.751	-26.68	77.07	30.6	70.2
July	ALD2	NJ_tox_2002	13	1.595	3.772	136.53	136.53	83.5	83.5
July	TOL	NJ_tox_2002	13	1.552	1.808	16.53	77.63	9.9	62
July	XYL	NJ_tox_2002	13	1.079	1.242	15.05	77.07	8.4	66.5
July	FORM	NJ_tox_2002	13	4.783	3.533	-26.14	40.79	-15.9	46.7

Table 4-28. Domain-wide summary of air toxics model performance evaluation results against 2002 PAMS data for July

Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
July	PAR	NJ_tox_2002	2190	26.73	75.41	182.12	208.02	115.00	118.90
July	OLE	NJ_tox_2002	2086	0.57	2.12	270.76	289.79	115.00	119.20
July	ALD2	NJ_tox_2002	1771	0.26	3.05	1077.99	1085.11	169.50	170.00
July	ISOP	NJ_tox_2002	1859	0.38	1.80	368.48	375.47	87.80	123.00
July	TOL	NJ_tox_2002	2188	0.62	1.73	180.46	208.73	108.30	114.00
July	XYL	NJ_tox_2002	2024	0.32	0.94	193.30	207.65	113.00	116.80
July	ETH	NJ_tox_2002	2012	0.82	1.53	87.41	108.25	73.80	83.00

*Site-specific results***Table 4-29. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for January at Camden Lab**

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Camden	January	PAR	NJ_tox_2002	4	8.36	107.70	1189.05	1189.05	170.80	170.80
Camden	January	OLE	NJ_tox_2002	4	1.50	2.83	89.21	89.21	76.20	76.20
Camden	January	ALD2	NJ_tox_2002	4	0.84	1.73	105.54	105.54	67.40	67.40
Camden	January	TOL	NJ_tox_2002	4	4.15	2.72	-34.41	103.68	42.20	112.20
Camden	January	XYL	NJ_tox_2002	4	0.86	1.79	108.22	108.22	70.30	70.30
Camden	January	FORM	NJ_tox_2002	4	1.45	1.53	4.97	29.59	4.10	28.50

Table 4-30. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for January at Rutgers University

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Rutgers	January	PAR	NJ_tox_2002	5	9.22	91.40	890.85	890.85	163.20	163.20
Rutgers	January	OLE	NJ_tox_2002	5	0.64	2.40	277.30	277.30	119.20	119.20
Rutgers	January	ALD2	NJ_tox_2002	5	0.90	1.40	54.78	54.78	42.70	42.70
Rutgers	January	TOL	NJ_tox_2002	5	3.40	2.54	-25.31	43.99	-11.30	41.20
Rutgers	January	XYL	NJ_tox_2002	5	2.39	1.65	-31.07	31.07	-37.60	37.60
Rutgers	January	FORM	NJ_tox_2002	5	1.93	1.20	-37.84	37.84	-41.10	41.10

Table 4-31. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for January at Chester

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Chester	January	PAR	NJ_tox_2002	5	4.80	63.55	1224.47	1224.47	169.50	169.50
Chester	January	OLE	NJ_tox_2002	5	0.32	1.90	496.98	496.98	139.70	139.70
Chester	January	ALD2	NJ_tox_2002	5	0.96	1.51	57.97	57.97	42.70	42.70
Chester	January	TOL	NJ_tox_2002	5	1.09	2.16	97.55	121.86	65.20	81.40
Chester	January	XYL	NJ_tox_2002	5	1.01	1.22	20.98	38.02	17.00	35.00
Chester	January	FORM	NJ_tox_2002	5	2.28	1.16	-49.06	49.06	-67.10	67.10

Table 4-32. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for January at Elizabeth

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Elizabeth	January	PAR	NJ_tox_2002	4	11.40	132.35	1061.22	1061.22	168.50	168.50
Elizabeth	January	OLE	NJ_tox_2002	4	4.41	3.54	-19.59	85.62	27.30	85.50
Elizabeth	January	ALD2	NJ_tox_2002	4	0.71	2.15	202.22	202.22	101.50	101.50
Elizabeth	January	TOL	NJ_tox_2002	4	1.32	5.21	295.65	295.65	120.00	120.00
Elizabeth	January	XYL	NJ_tox_2002	4	0.77	3.34	333.18	333.18	126.30	126.30
Elizabeth	January	FORM	NJ_tox_2002	4	1.38	1.82	32.14	32.14	28.30	28.30

Table 4-33. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for July at Camden Lab

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Camden	July	PAR	NJ_tox_2002	4	6.41	83.79	1208.12	1208.12	169.70	169.70
Camden	July	OLE	NJ_tox_2002	4	1.16	1.66	42.77	48.92	37.50	42.40
Camden	July	ALD2	NJ_tox_2002	4	0.81	3.36	316.59	316.59	123.90	123.90
Camden	July	TOL	NJ_tox_2002	4	1.79	1.15	-35.78	60.44	-14.80	53.30
Camden	July	XYL	NJ_tox_2002	4	0.69	0.86	23.85	40.50	20.20	36.20
Camden	July	FORM	NJ_tox_2002	4	2.40	3.30	37.47	57.51	42.30	57.90

Table 4-34. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for July at Rutgers University

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Rutgers	July	PAR	NJ_tox_2002	2	7.85	69.10	780.75	780.75	155.80	155.80
Rutgers	July	OLE	NJ_tox_2002	2	0.60	1.42	136.92	136.92	80.40	80.40
Rutgers	July	ALD2	NJ_tox_2002	2	2.21	3.97	79.46	79.46	53.70	53.70
Rutgers	July	TOL	NJ_tox_2002	2	1.31	0.87	-33.15	33.15	-39.20	39.20
Rutgers	July	XYL	NJ_tox_2002	2	1.44	0.63	-56.62	56.62	-79.70	79.70
Rutgers	July	FORM	NJ_tox_2002	2	6.60	3.81	-42.28	42.28	-54.70	54.70

Table 4-35. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for July at Chester

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Chester	July	PAR	NJ_tox_2002	2	6.43	62.60	874.32	874.32	158.20	158.20
Chester	July	OLE	NJ_tox_2002	2	0.52	1.38	167.09	167.09	90.80	90.80
Chester	July	ALD2	NJ_tox_2002	2	2.42	3.79	56.94	56.94	40.80	40.80
Chester	July	TOL	NJ_tox_2002	2	1.63	0.74	-54.47	60.09	-56.80	67.40
Chester	July	XYL	NJ_tox_2002	2	1.75	0.50	-71.36	71.36	-92.90	92.90
Chester	July	FORM	NJ_tox_2002	2	9.36	3.72	-60.29	60.29	-87.40	87.40

Table 4-36. Summary of air toxics model performance evaluation results against 2002 NJ Air Toxics data for July at Elizabeth

Site	Month	CB4 species	Model	N	Obs. mean	Mod. mean	NMB	%NME	%MFB	%MFE
Elizabeth	July	PAR	NJ_tox_2002	5	10.05	121.90	1112.94	1112.94	169.40	169.40
Elizabeth	July	OLE	NJ_tox_2002	5	4.83	2.11	-56.44	75.68	-18.80	80.10
Elizabeth	July	ALD2	NJ_tox_2002	5	1.65	4.01	143.20	143.20	80.30	80.30
Elizabeth	July	TOL	NJ_tox_2002	5	1.43	3.14	118.90	118.90	76.10	76.10
Elizabeth	July	XYL	NJ_tox_2002	5	0.98	2.10	113.76	113.76	74.80	74.80
Elizabeth	July	FORM	NJ_tox_2002	5	4.14	3.54	-14.43	14.43	-18.20	18.20

Box plots

Box plots showing average monthly concentrations at NJ Air Toxics network sites and average predicted monthly concentrations are presented in Figure 4-27 through Figure 4-32. It is more difficult to determine whether observed seasonal patterns are well reproduced by the model because only two months were modeled. However, as seen in

the analysis of the regional haze model, July values were generally on par with other summertime values, as were January values with other wintertime values. Therefore, we can examine the seasonal variation using these representative months. Results indicate that the air toxics model generally overestimates values in both summer and winter months, but observations better match predictions in the summer. The box plots show first and third quartile values as the bottom and top of the boxes, respectively, and the central line represents the median value for both observed and predicted concentrations.

The air toxics model uniformly overpredicts by a factor of approximately two for higher aldehydes, as shown in Figure 4-27, with the model reproducing the observed higher summertime concentrations and lower wintertime concentrations pattern. The model reproduces concentration levels in both summer and winter for formaldehyde, as shown in Figure 4-28. Olefins and toluene are overpredicted by a factor of three in the winter, but well-reproduced in the summer (Figure 4-29 and Figure 4-31, respectively). Levels of xylenes match well for both summer and winter months, as shown in Figure 4-32. Levels of paraffins are grossly overpredicted in both summer and winter (Figure 4-30).

Average diurnal trends (July only) from the PAMS network are compared against model predictions in box plots in Figure 4-33 through Figure 4-39. Similar to the seasonal box plots, these plots show that while observed CB4 species patterns are generally well reproduced in the model, the levels are often grossly overpredicted. Modeled diurnal profiles are similar for ethylene, olefins, paraffins, toluene, and xylenes; high nighttime levels with daytime lows due to photochemistry. While the pattern is present in observations, levels match well only for ethylene, toluene, and xylenes (generally overpredicted by a factor of two or less), as shown in Figure 4-34, Figure 4-38, and Figure 4-39, respectively. Olefins (Figure 4-36) are grossly overpredicted at night, and paraffins (Figure 4-37) are universally overpredicted. The modeled diurnal profile for isoprene matches the observed profile except in the late evening, when the model shows a peak while the observations show a general tailing off of levels, as shown in Figure 4-35. Acetaldehyde and higher aldehydes are universally overpredicted at all hours of the day (Figure 4-33).

Figure 4-27. Comparison of monthly average higher aldehyde concentrations for the NJ Air Toxics network at all sites for the air toxics model

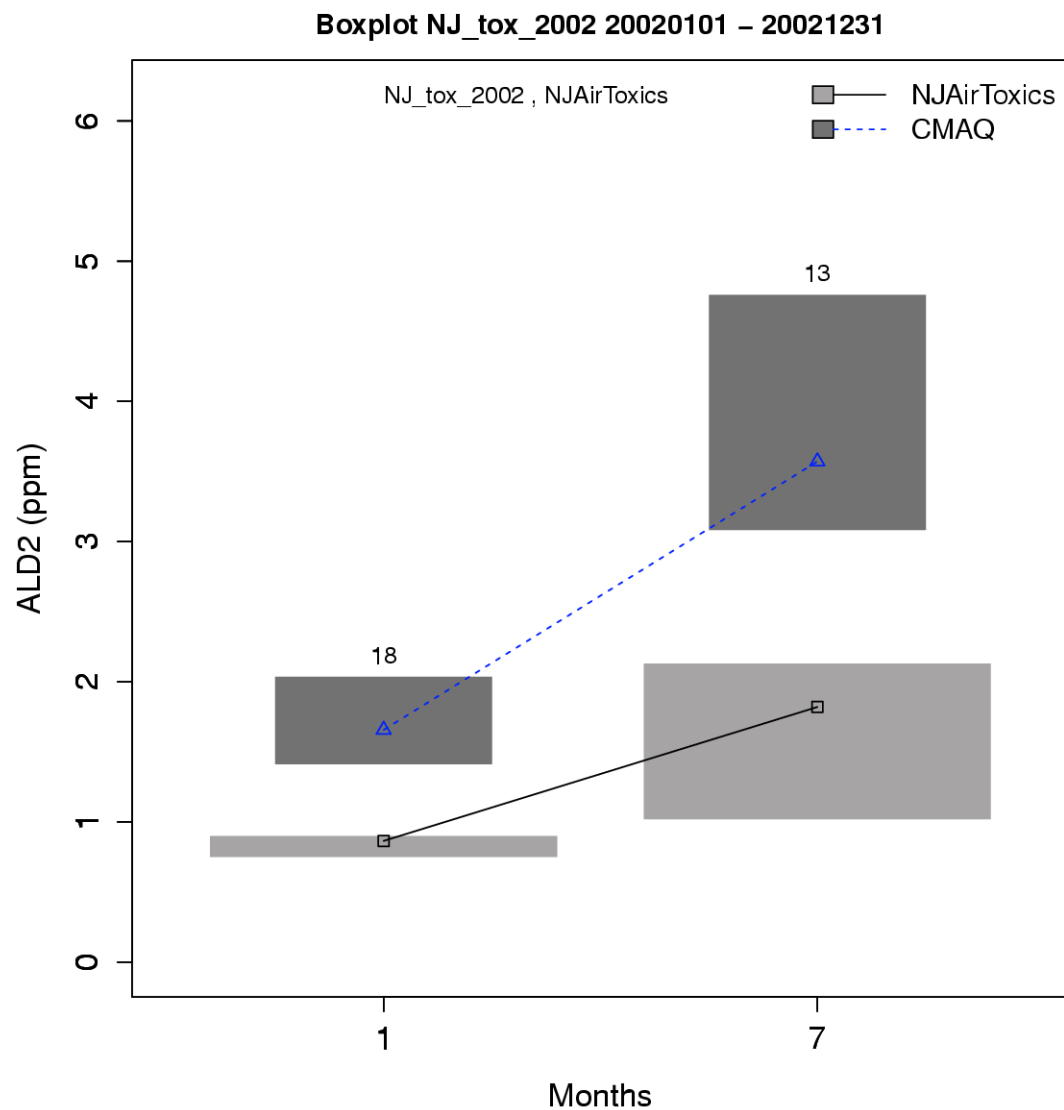


Figure 4-28. Comparison of monthly average formaldehyde concentrations for the NJ Air Toxics network at all sites for the air toxics model

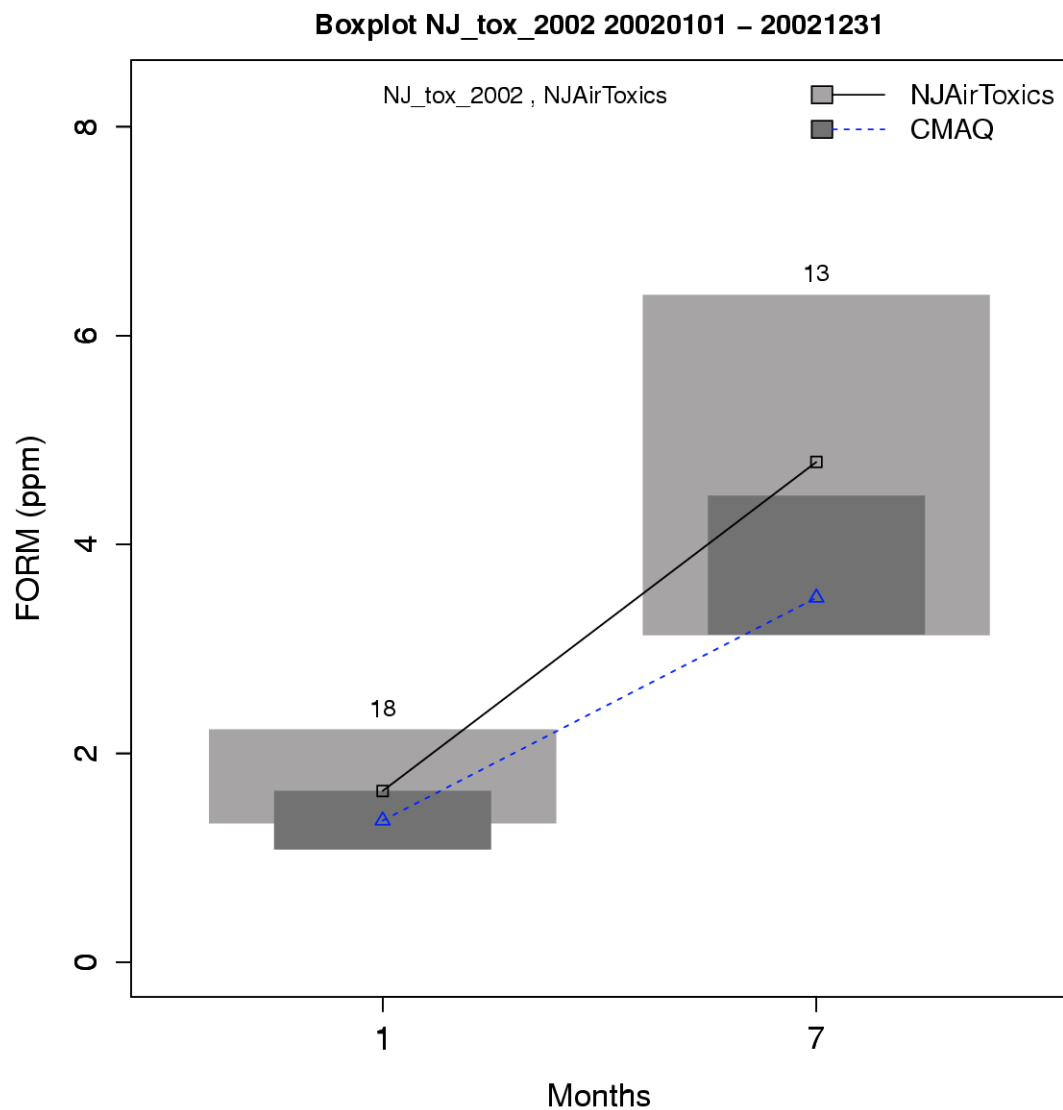


Figure 4-29. Comparison of monthly average olefin concentrations for the NJ Air Toxics network at all sites for the air toxics model

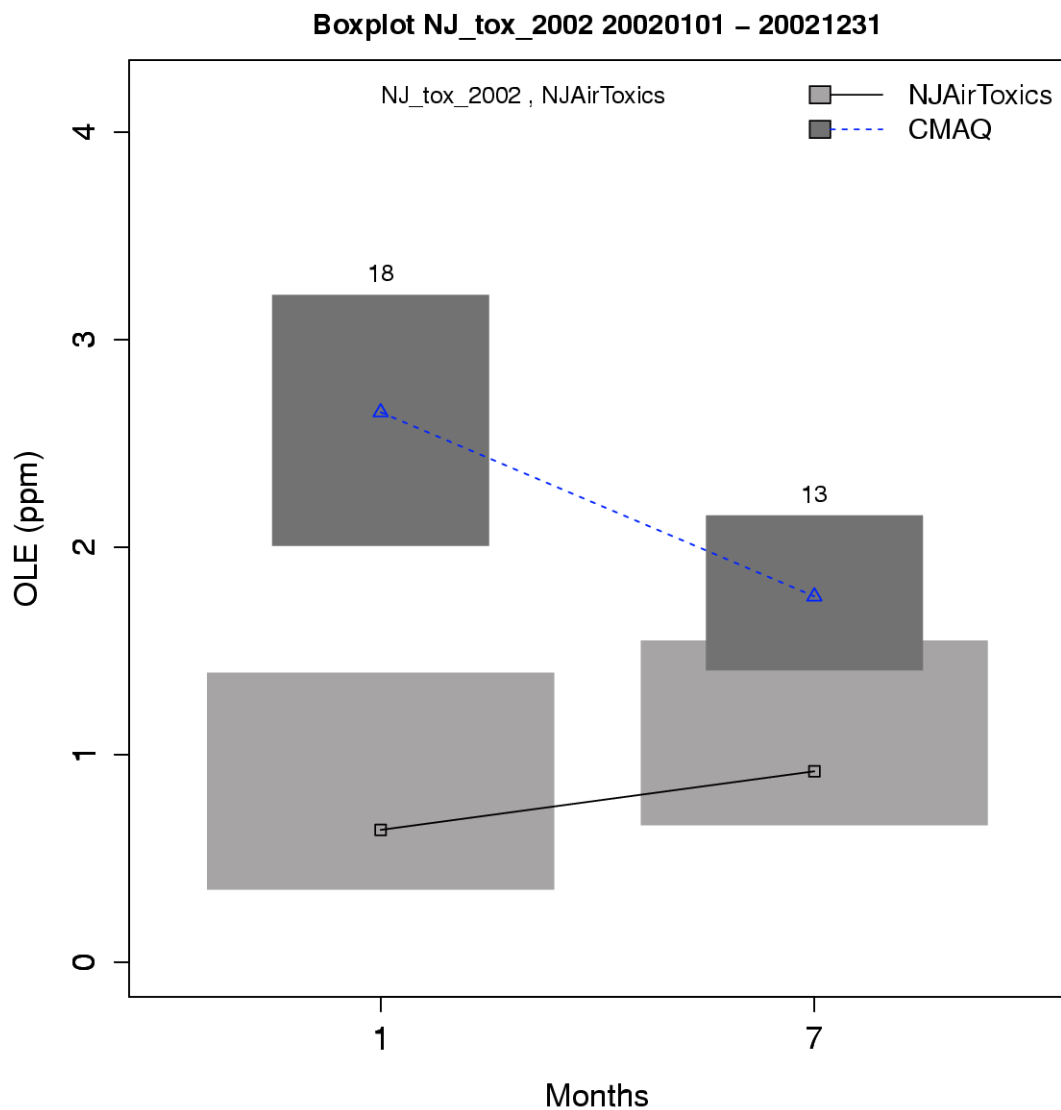


Figure 4-30. Comparison of monthly average paraffin concentrations for the NJ Air Toxics network at all sites for the air toxics model

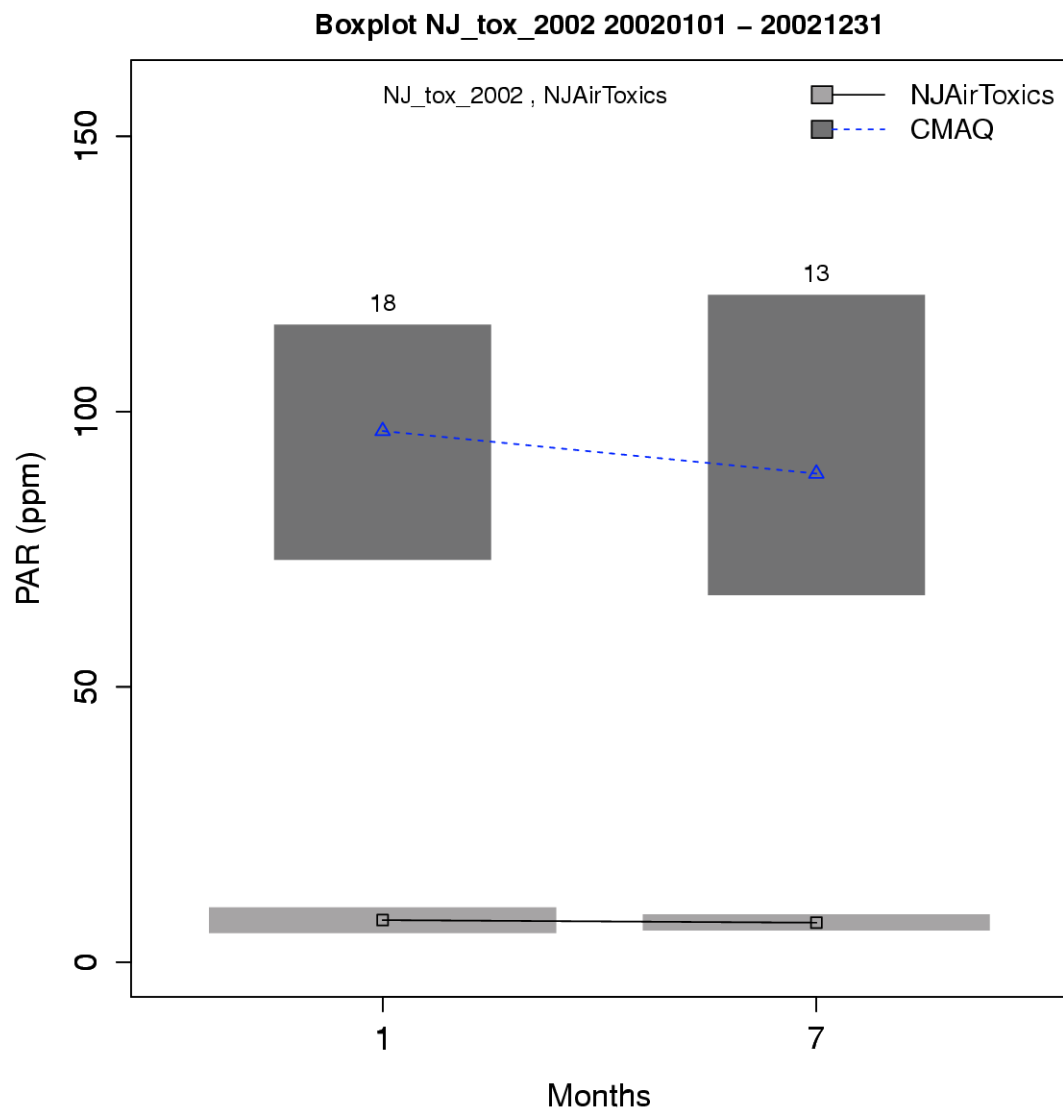


Figure 4-31. Comparison of monthly average toluene concentrations for the NJ Air Toxics network at all sites for the air toxics model

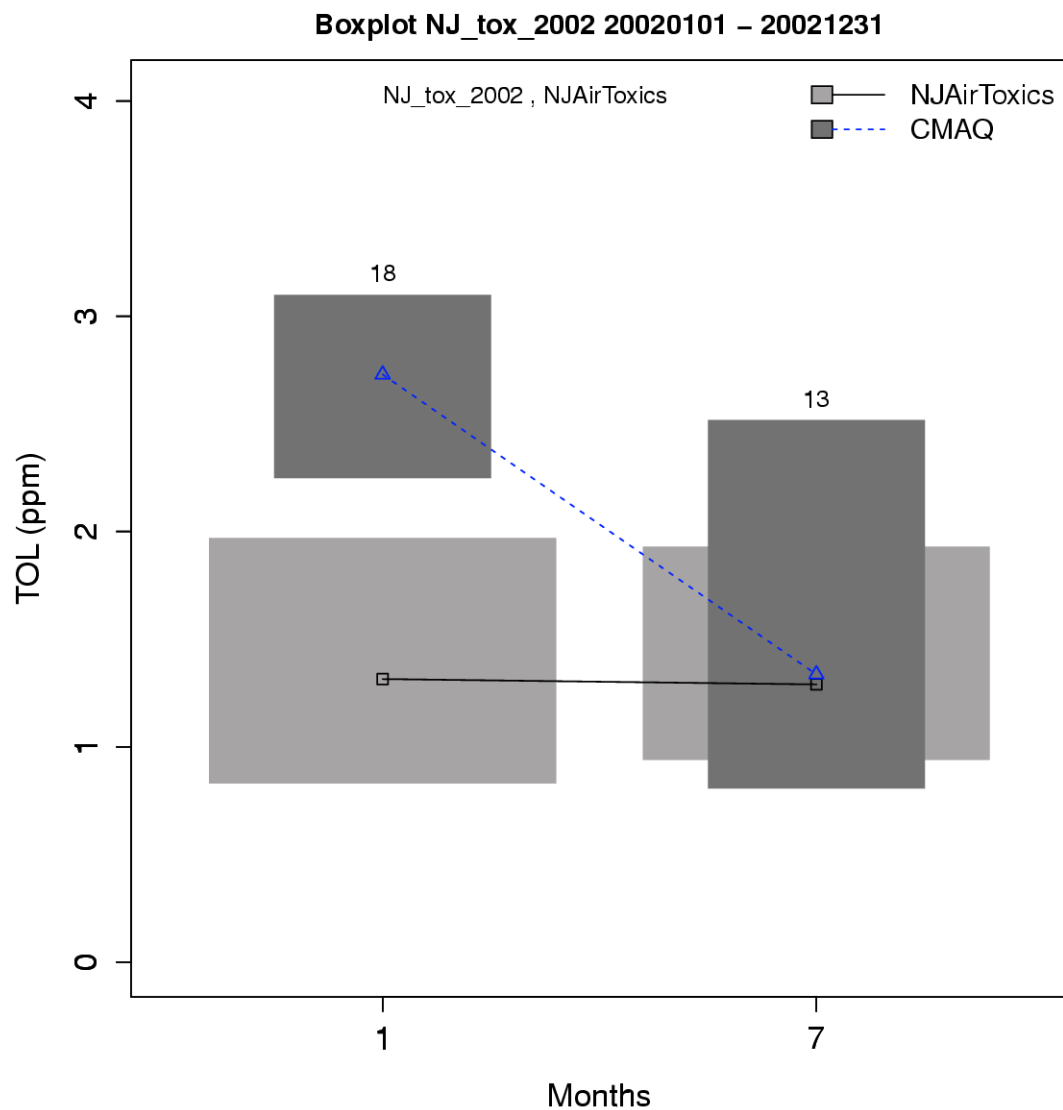


Figure 4-32. Comparison of monthly average xylenes concentrations for the NJ Air Toxics network at all sites for the air toxics model

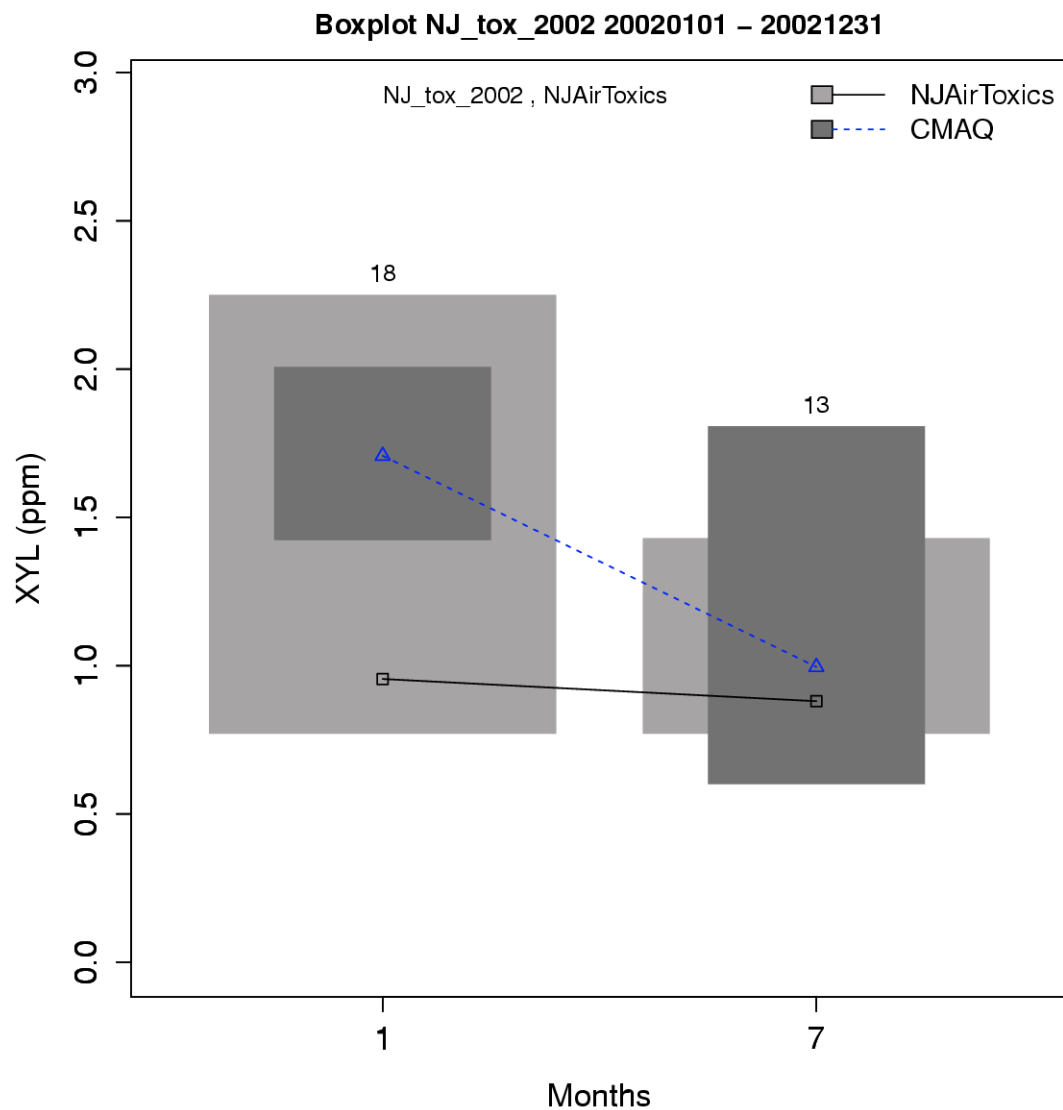


Figure 4-33. Comparison of July diurnal patterns for higher aldehydes from the PAMS network at all sites for the air toxics model

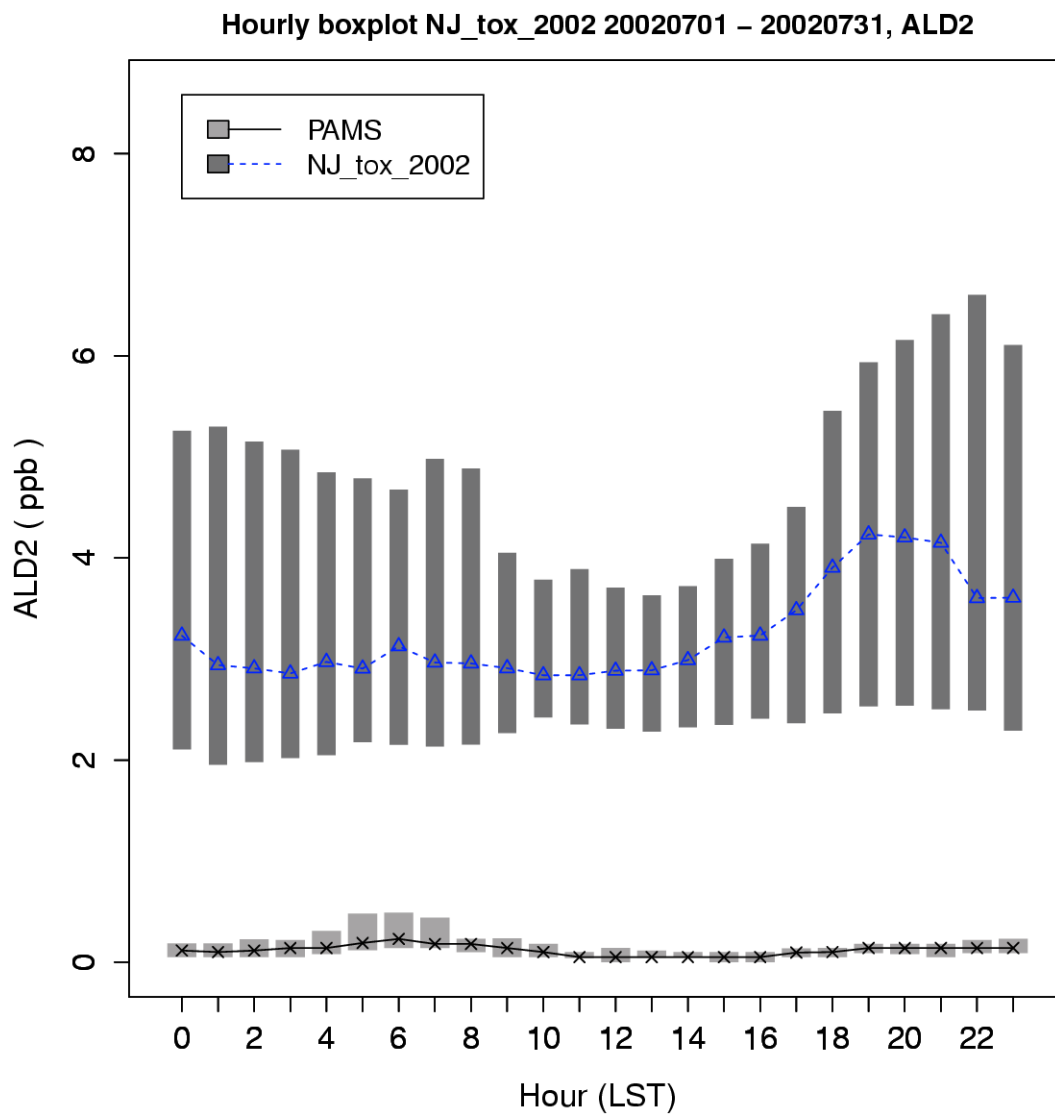


Figure 4-34. Comparison of July diurnal patterns for ethylene from the PAMS network at all sites for the air toxics model

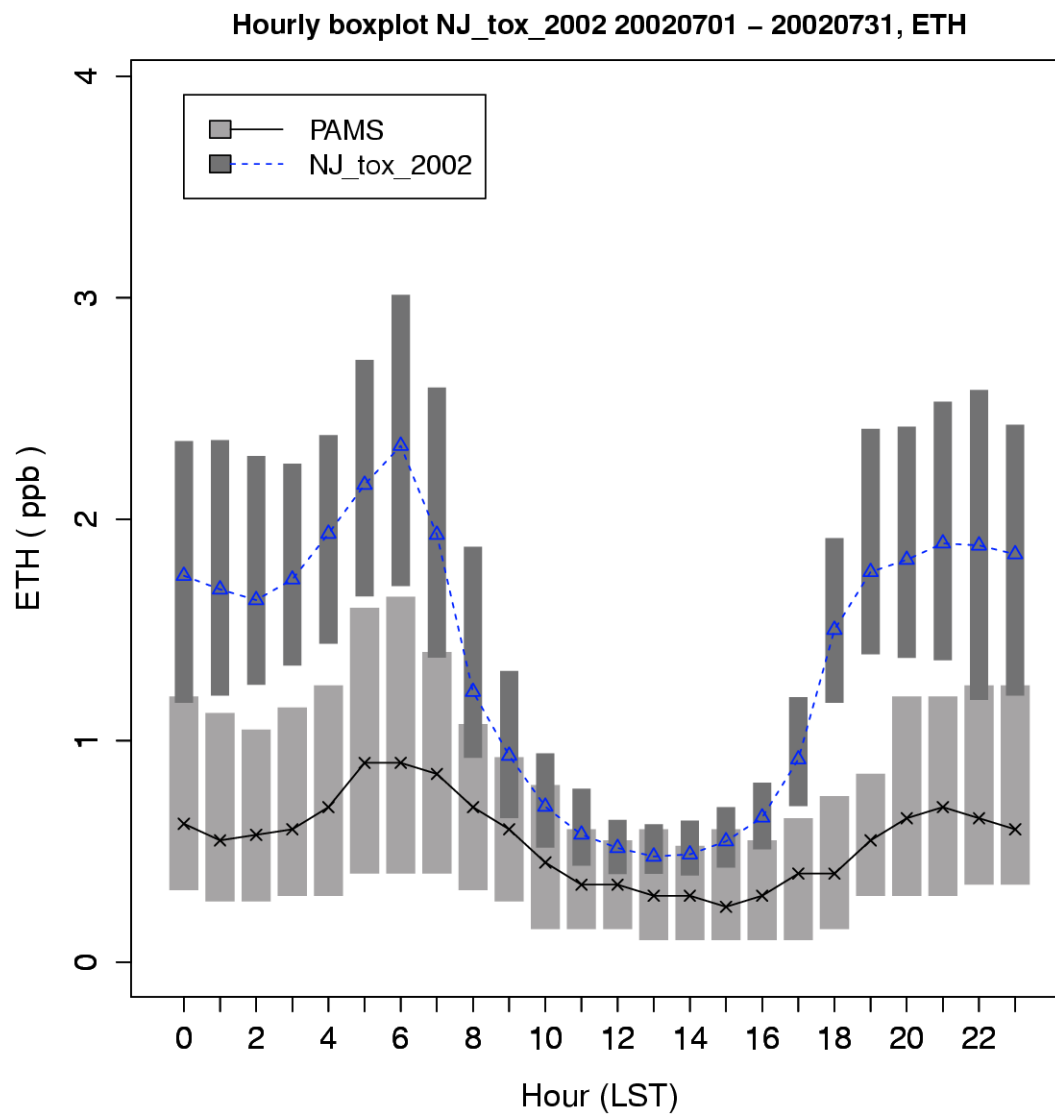


Figure 4-35. Comparison of July diurnal patterns for isoprene from the PAMS network at all sites for the air toxics model

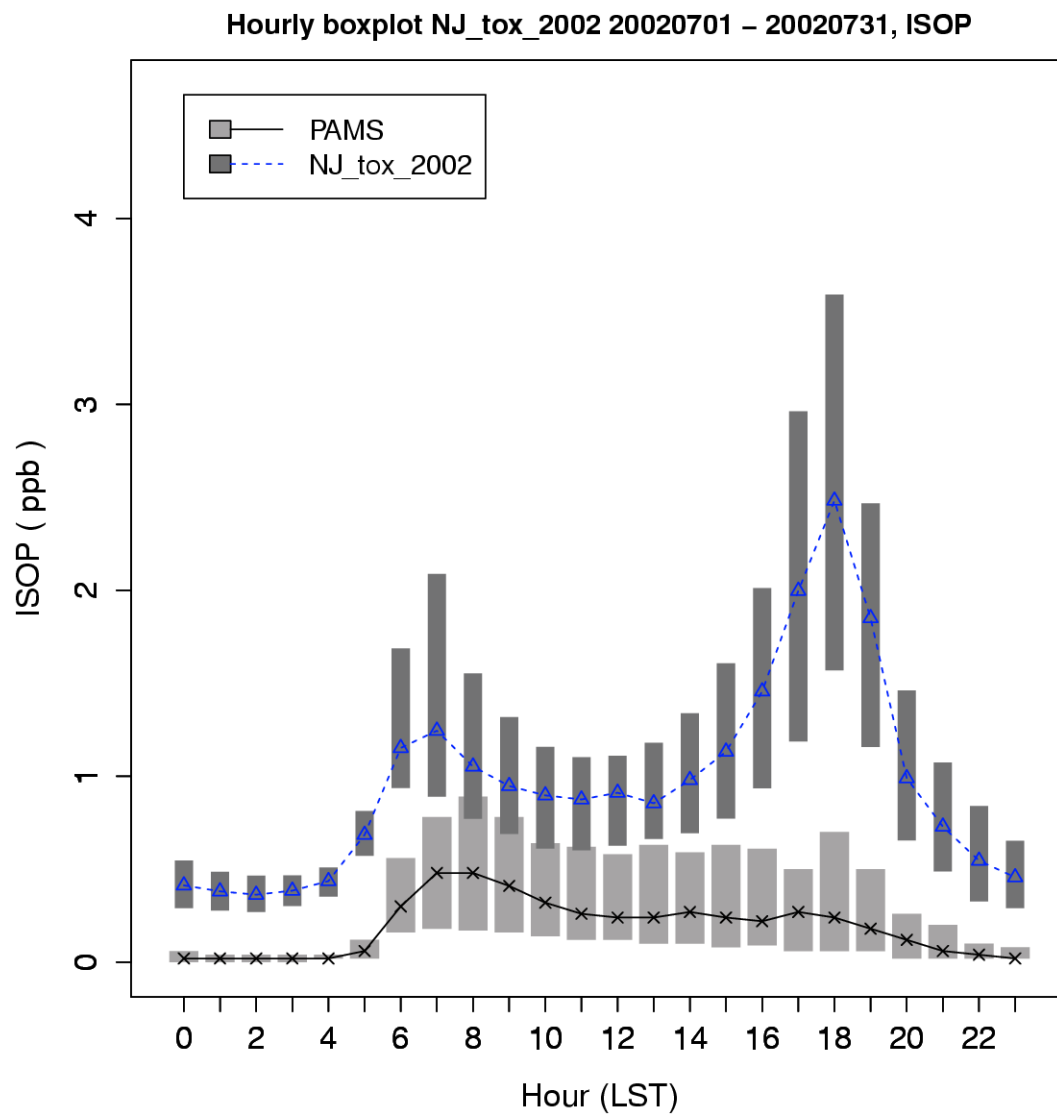


Figure 4-36. Comparison of July diurnal patterns for olefins from the PAMS network at all sites for the air toxics model

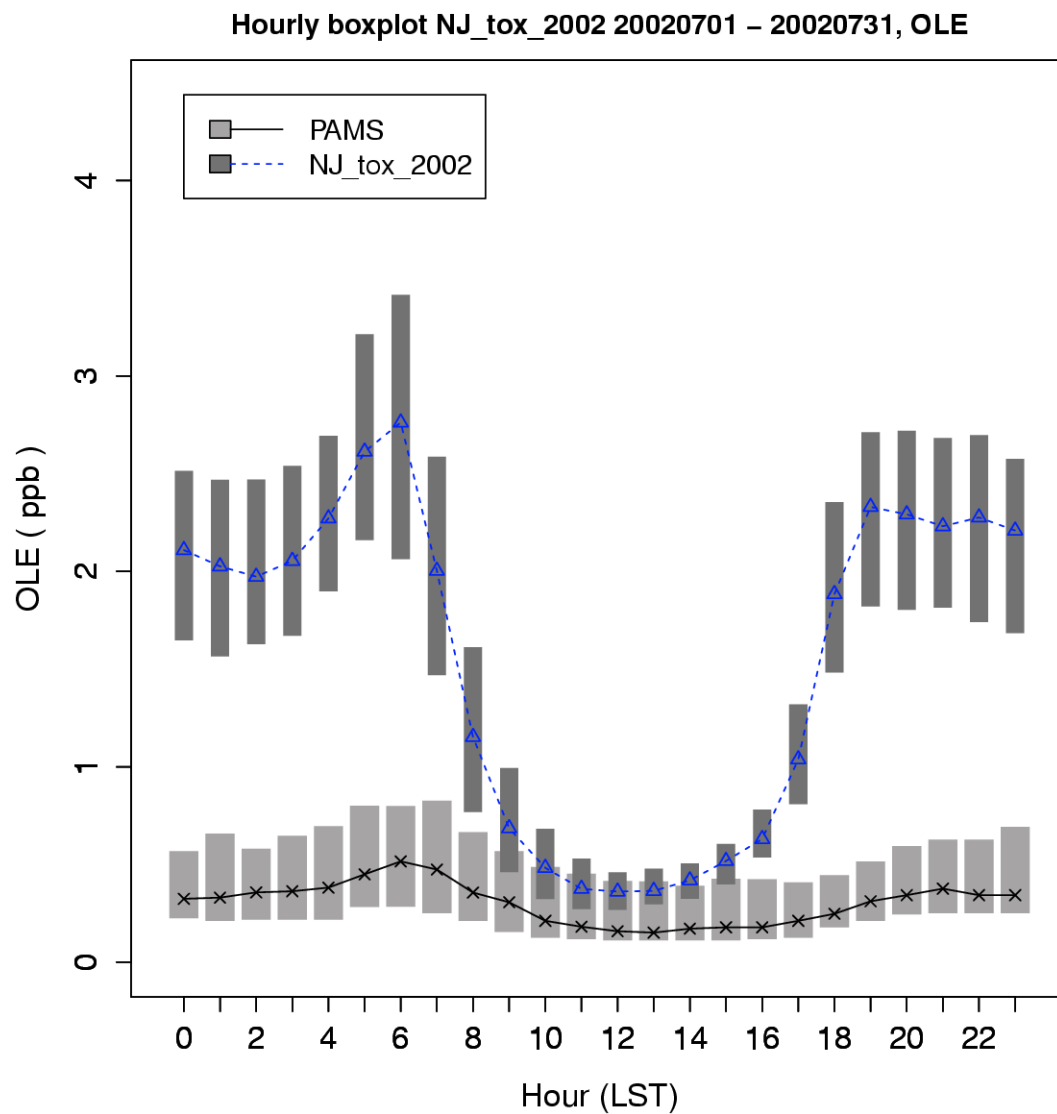


Figure 4-37. Comparison of July diurnal patterns for paraffins from the PAMS network at all sites for the air toxics model

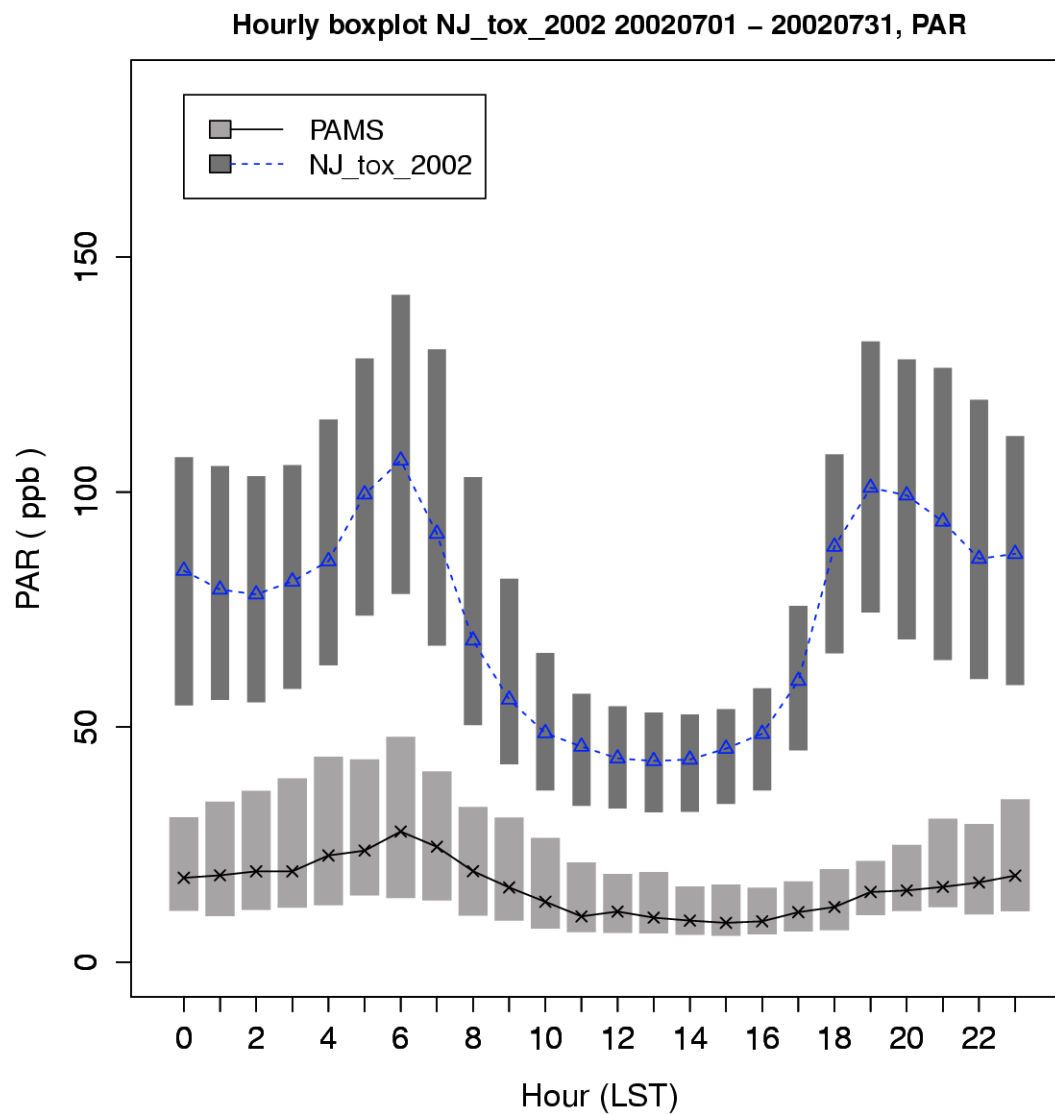


Figure 4-38. Comparison of July diurnal patterns for toluene from the PAMS network at all sites for the air toxics model

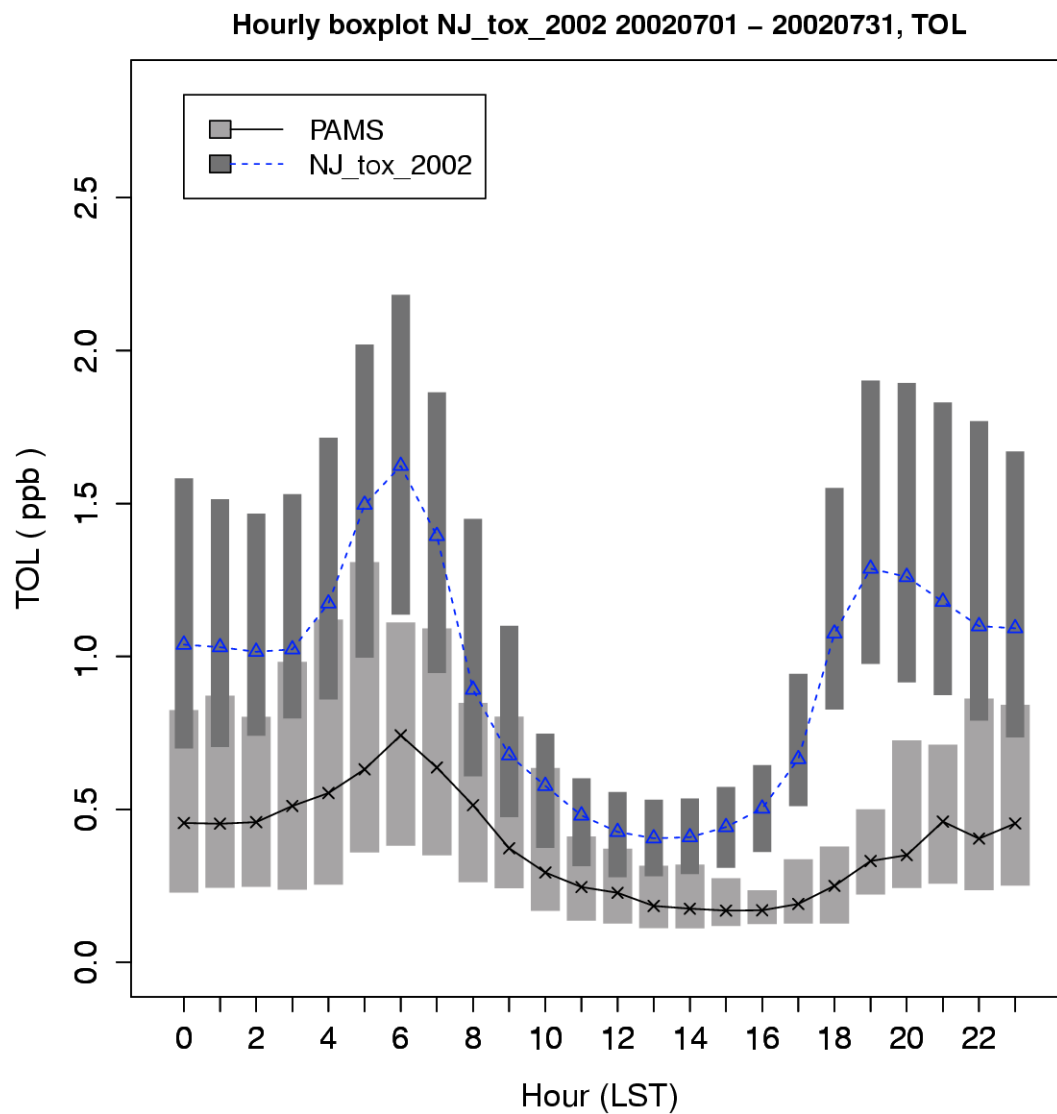
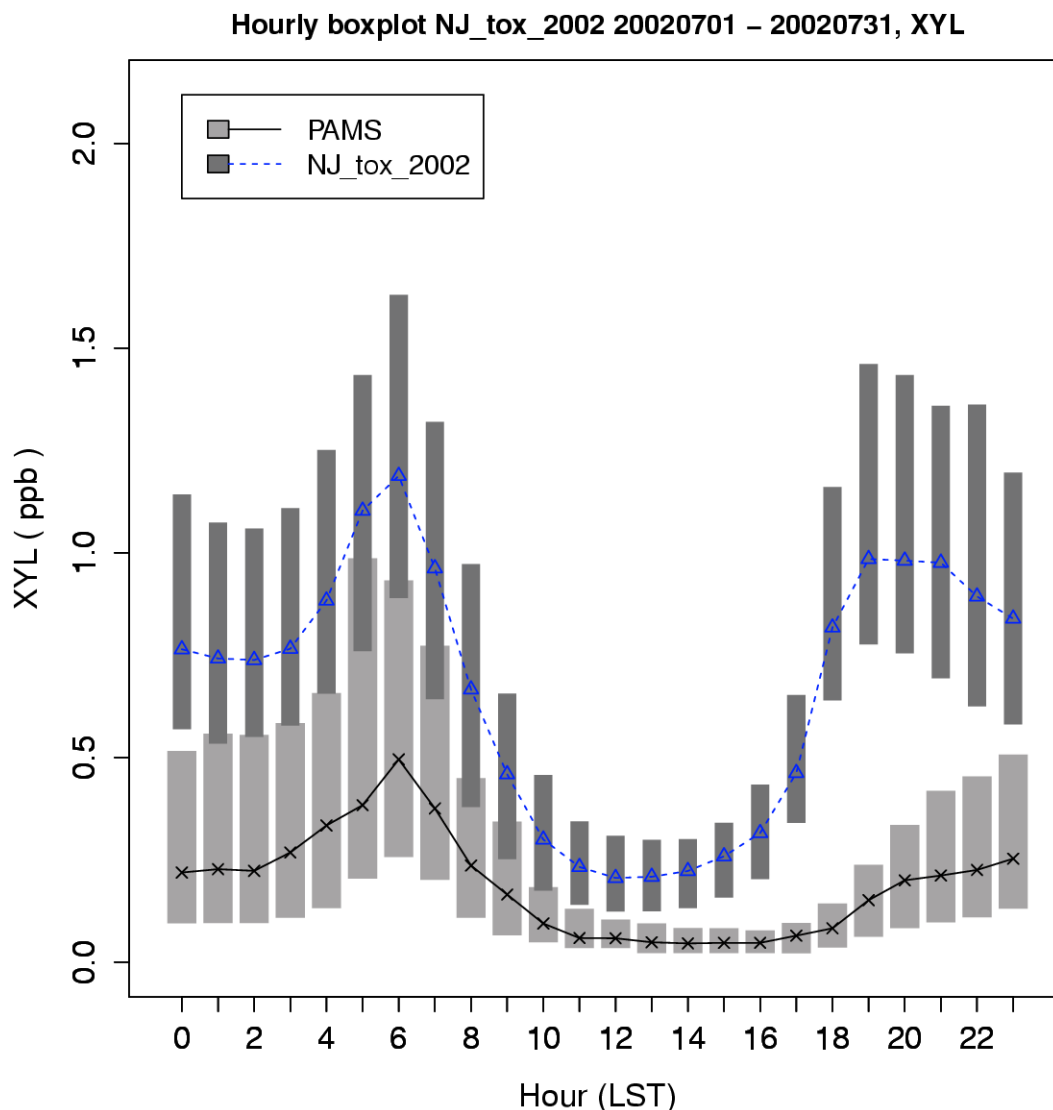


Figure 4-39. Comparison of July diurnal patterns for xylenes from the PAMS network at all sites for the air toxics model



Scatter plots

Results of scatter plots are shown for January and July for the NJ Air Toxics network. Data are paired in space and time.

Scatter plots for the winter show reasonable agreement with some overprediction for the model against observed concentrations. Aldehydes (shown in Figure 4-40) appear to have similar overpredictions at high and low observed concentrations, with some predictions agreeing reasonably well with observations. Formaldehyde predictions also match observations reasonably well, though the scatter is wide (see Figure 4-41). Olefins are generally well predicted (though somewhat overpredicted at lower concentration levels), but the model failed to predict the highest observed concentration, which greatly affected bias and error metrics (see Figure 4-42). Paraffins are almost universally

overpredicted; observed levels rarely exceed 20 ppb while modeled levels are often in excess of 100 ppb (Figure 4-43). Performance for xylenes is similar to acetaldehyde, with significant overprediction but adequate performance at points (Figure 4-45). Toluene (Figure 4-44) is more like formaldehyde, with lower concentrations achieving better predictions than higher concentrations, which appear to be significantly underestimated. Such large underestimates may be the result of a gap in emission inventories.

Summertime scatter plots paired in space and time (Figure 4-46 through Figure 4-51) show different patterns of agreement between observations and predictions than the winter scatter plots do for some species, and similar patterns for others. Levels of higher aldehydes (Figure 4-46) are overpredicted consistently, with considerably more error than during the summer month. Unlike in the winter, formaldehyde (Figure 4-47) is well represented in the model, though the model does not reproduce high observed values well. Summertime olefin values (Figure 4-48) are well represented in the model at lower concentration levels, and poorly reproduced at higher observed levels, when the model fails to reproduce high concentration events. As in the winter, summer levels of paraffins are grossly overpredicted (Figure 4-49). Levels of toluene (Figure 4-50) and xylenes (Figure 4-51) are often overpredicted, and higher observed values tend to the highest scatter (i.e., error).

Figure 4-40. Comparison of January scatter plot for higher aldehydes from NJ Air Toxics sites for the air toxics model

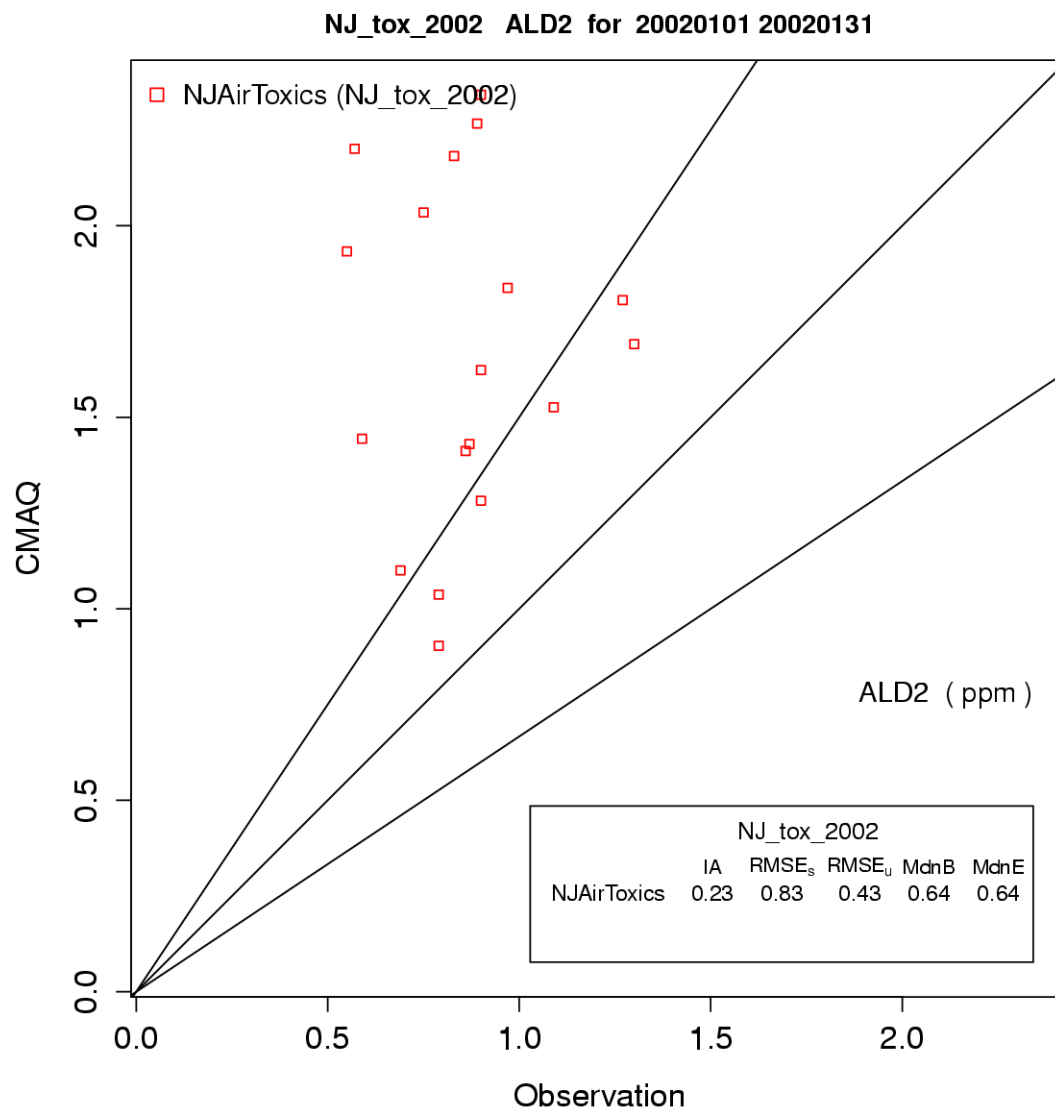


Figure 4-41. Comparison of January scatter plot for formaldehyde from NJ Air Toxics sites for the air toxics model

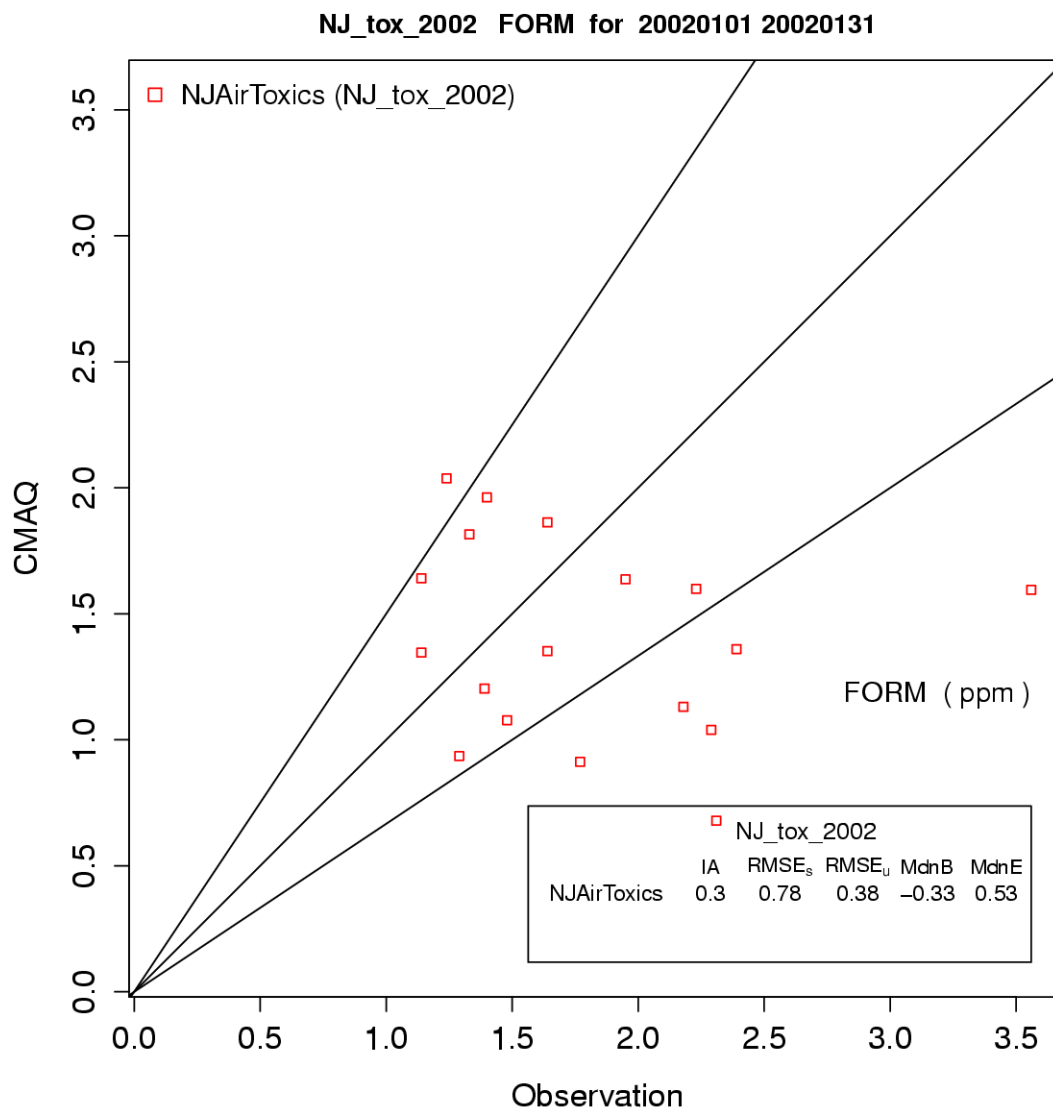


Figure 4-42. Comparison of January scatter plot for olefins from NJ Air Toxics sites for the air toxics model

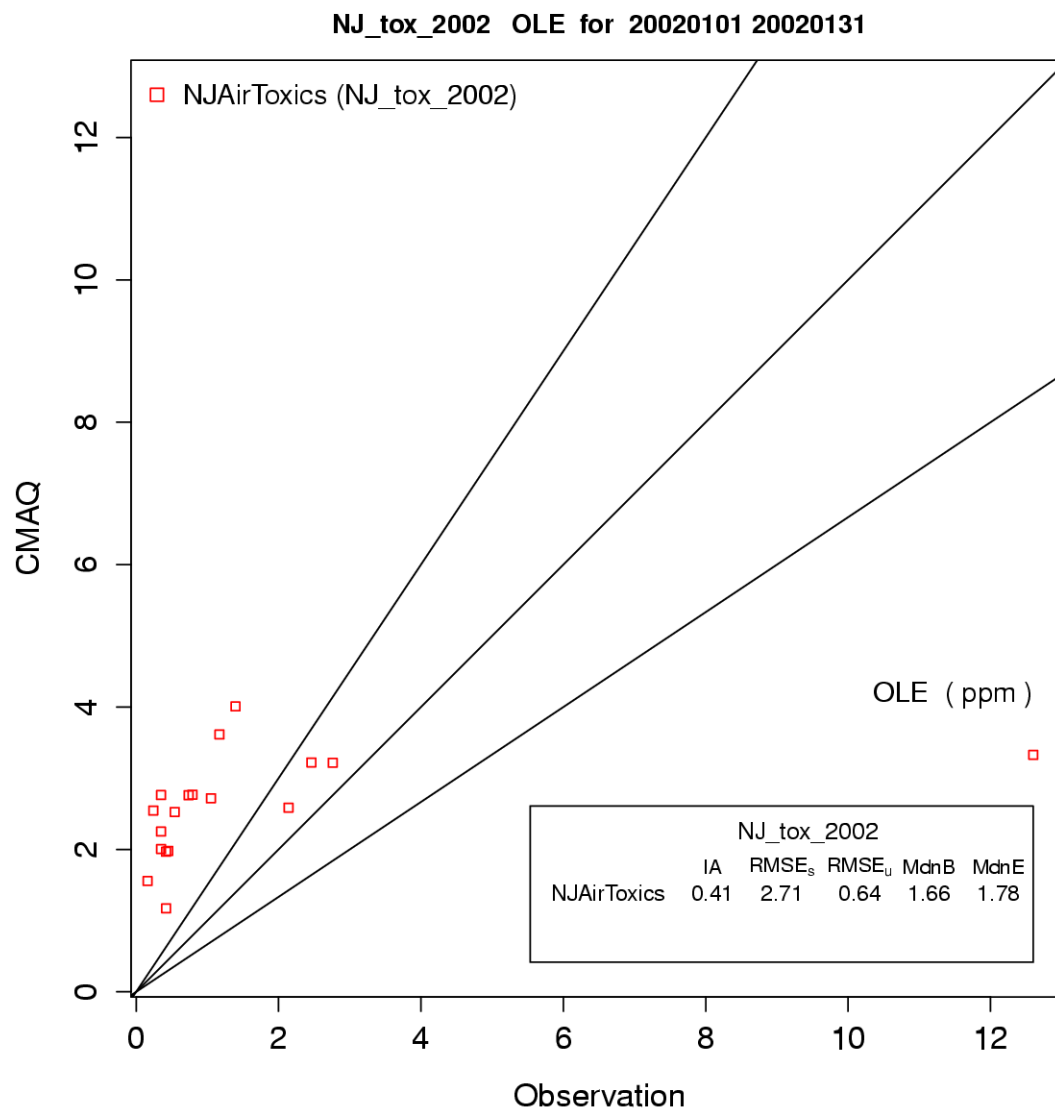


Figure 4-43. Comparison of January scatter plot for paraffins from NJ Air Toxics sites for the air toxics model

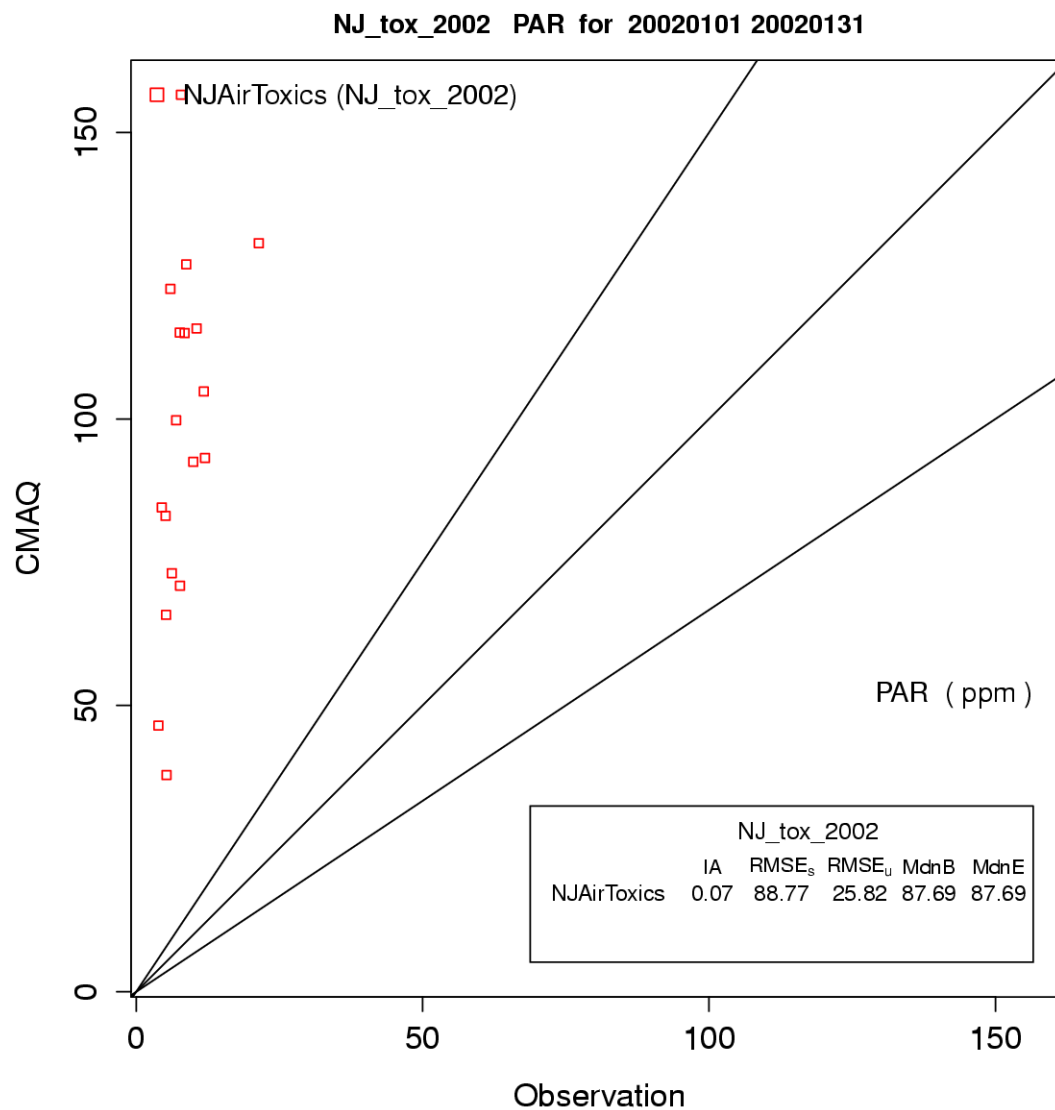


Figure 4-44. Comparison of January scatter plot for toluene from NJ Air Toxics sites for the air toxics model

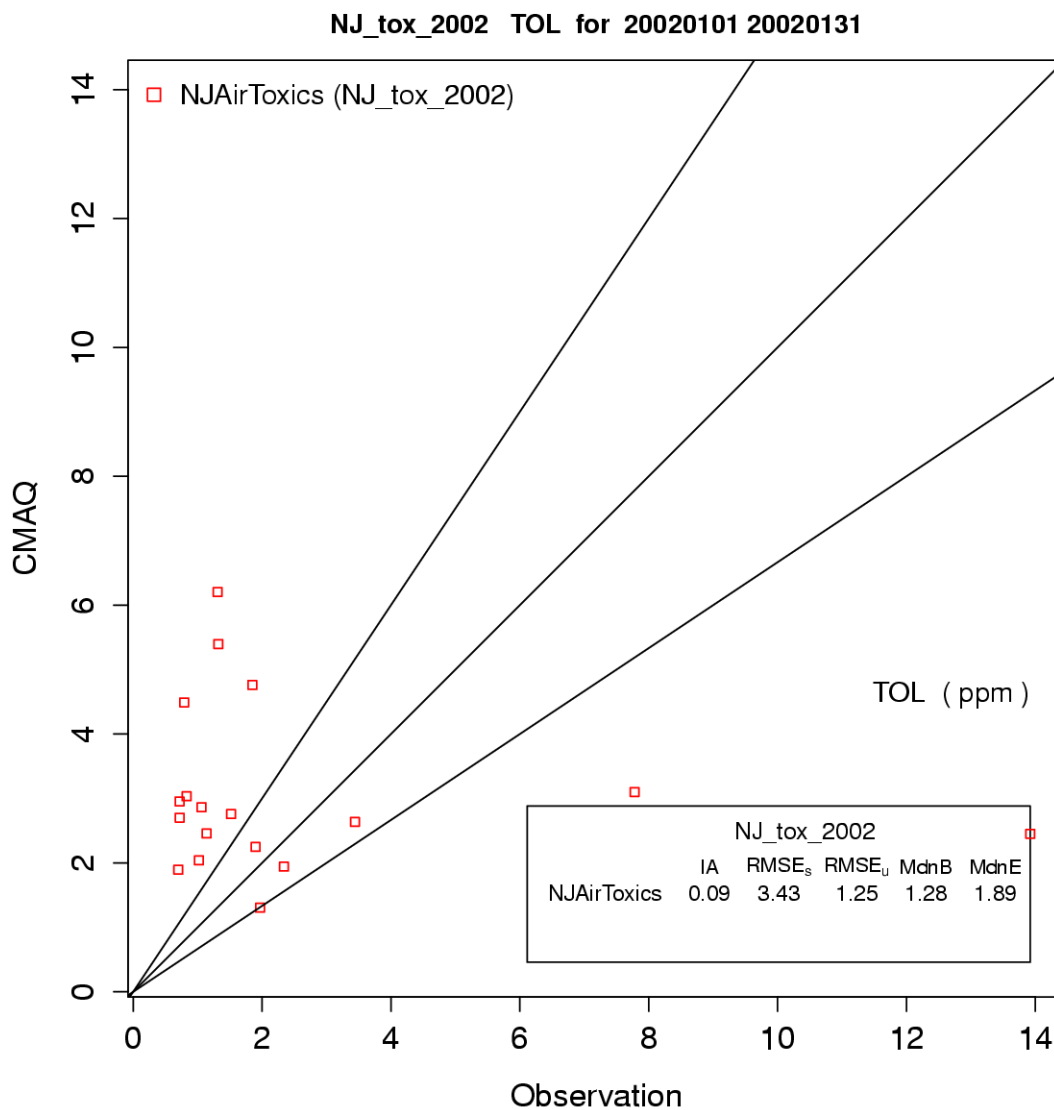


Figure 4-45. Comparison of January scatter plot for xylenes from NJ Air Toxics sites for the air toxics model

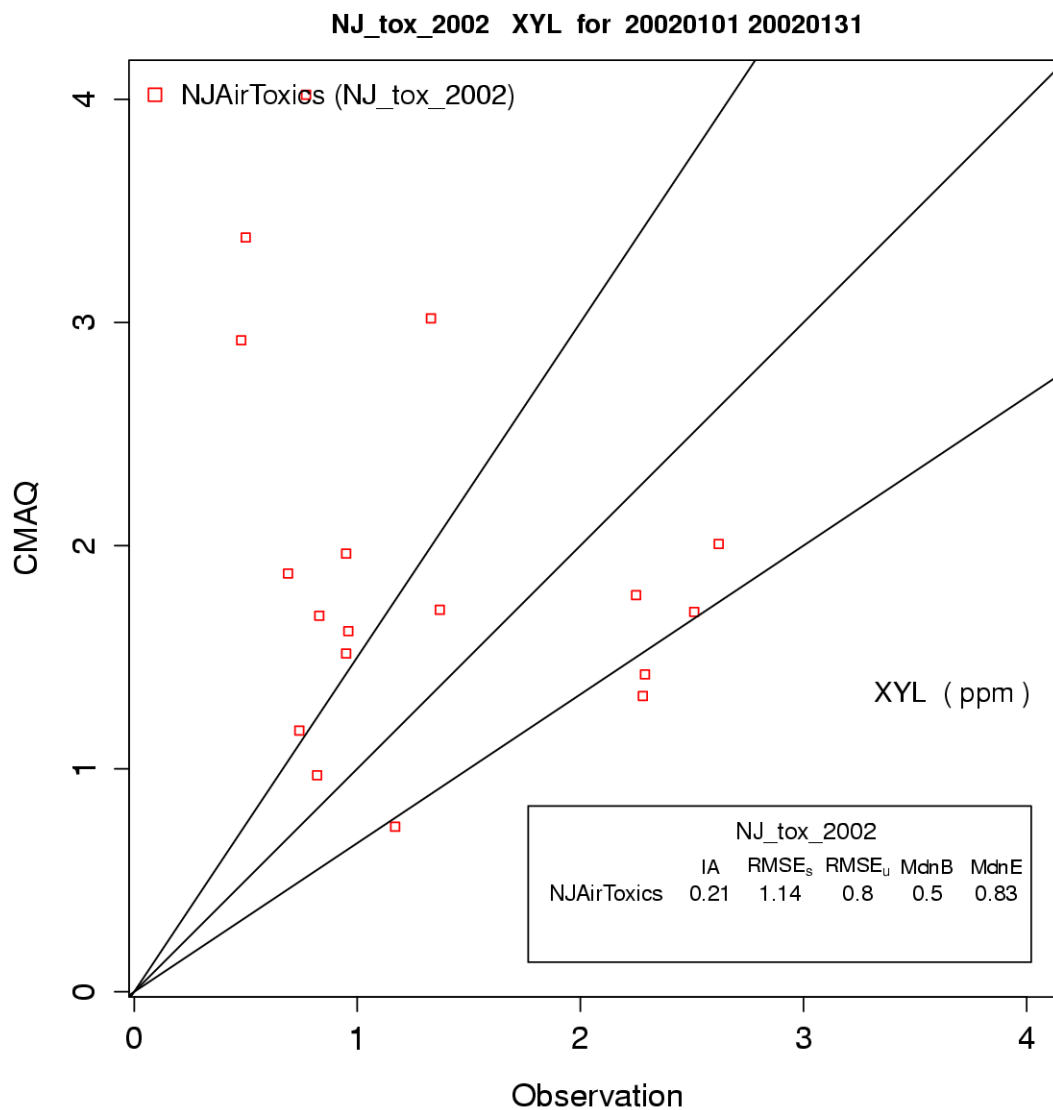


Figure 4-46. Comparison of July scatter plot for higher aldehydes from NJ Air Toxics sites for the air toxics model

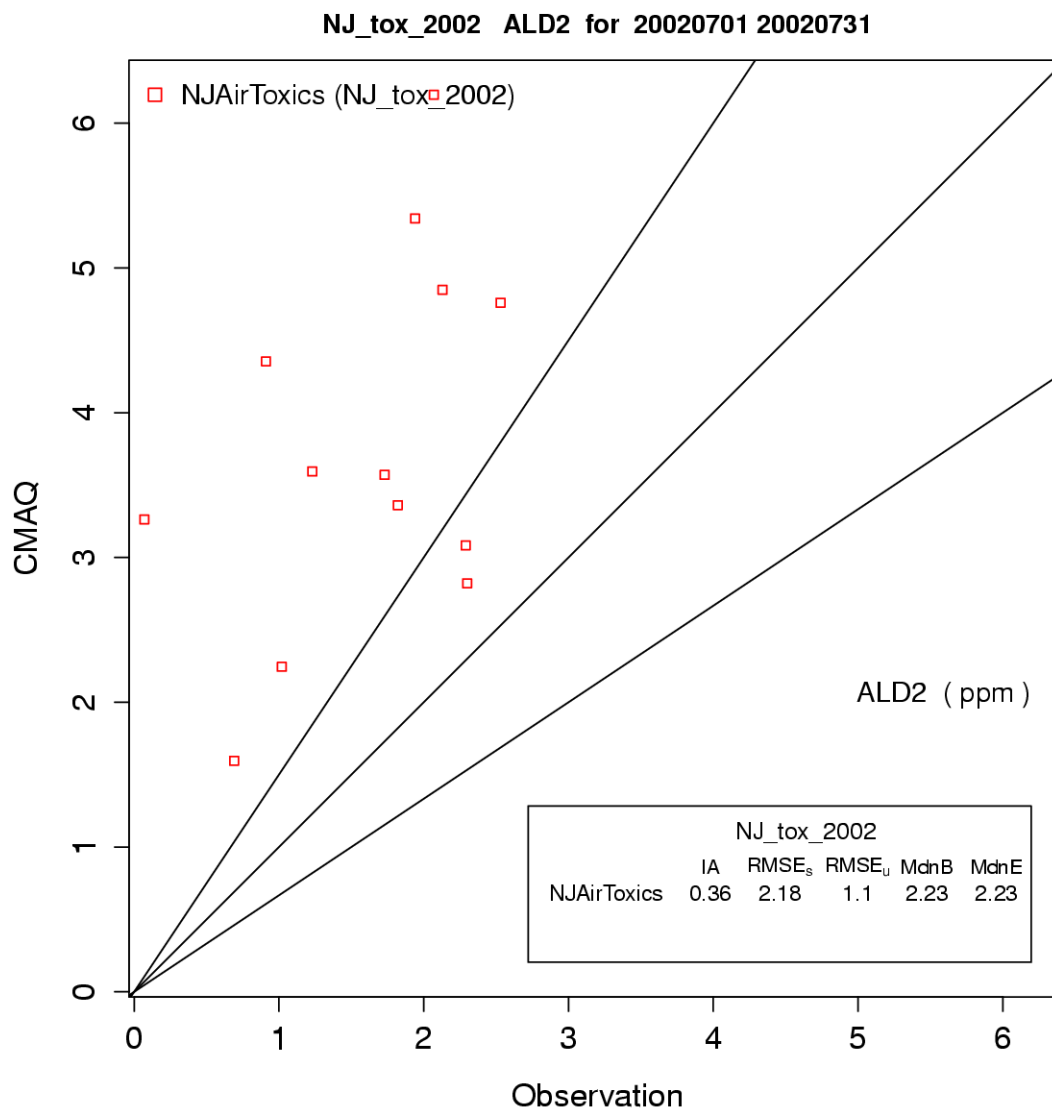


Figure 4-47. Comparison of July scatter plot for formaldehyde from NJ Air Toxics sites for the air toxics model

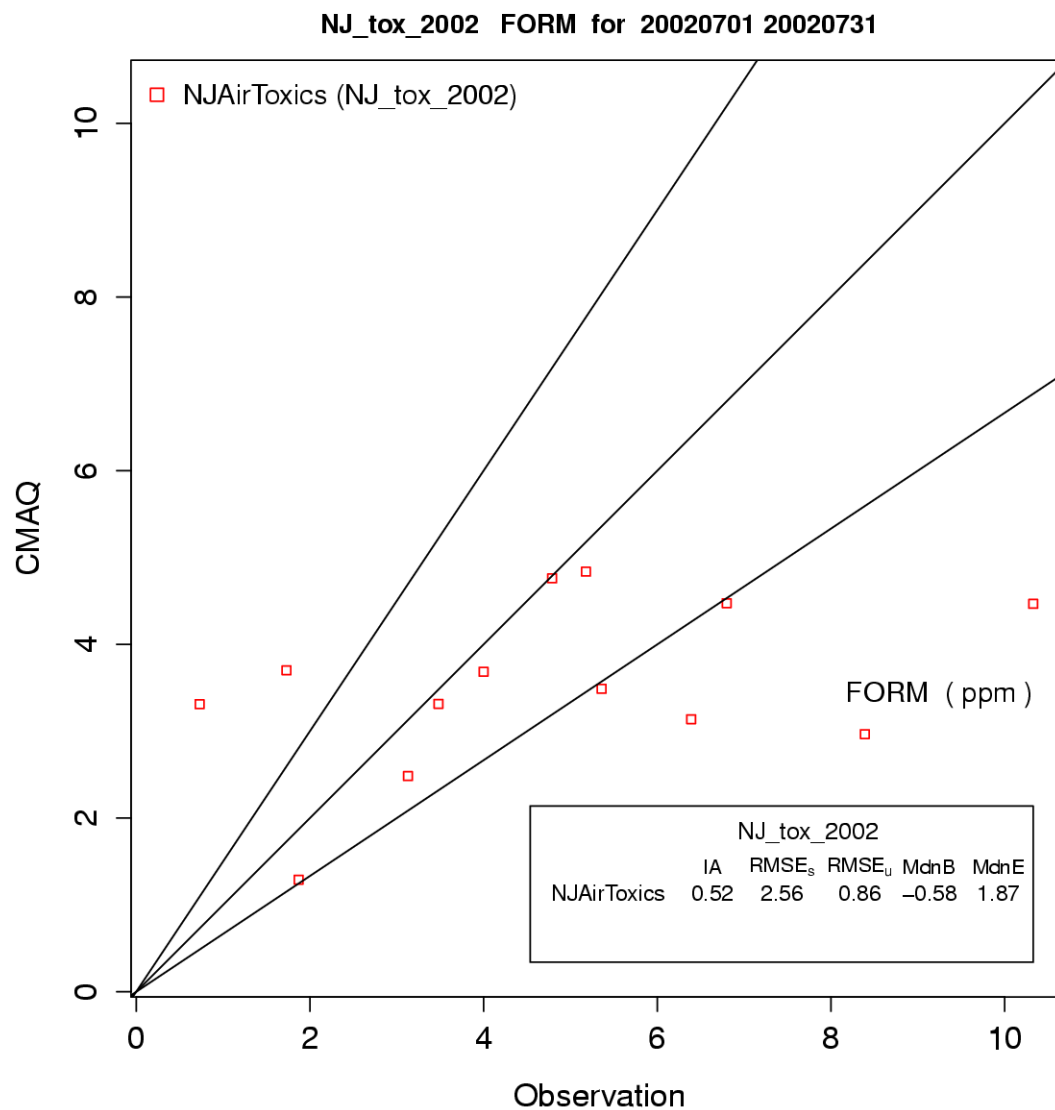


Figure 4-48. Comparison of July scatter plot for olefins from NJ Air Toxics sites for the air toxics model

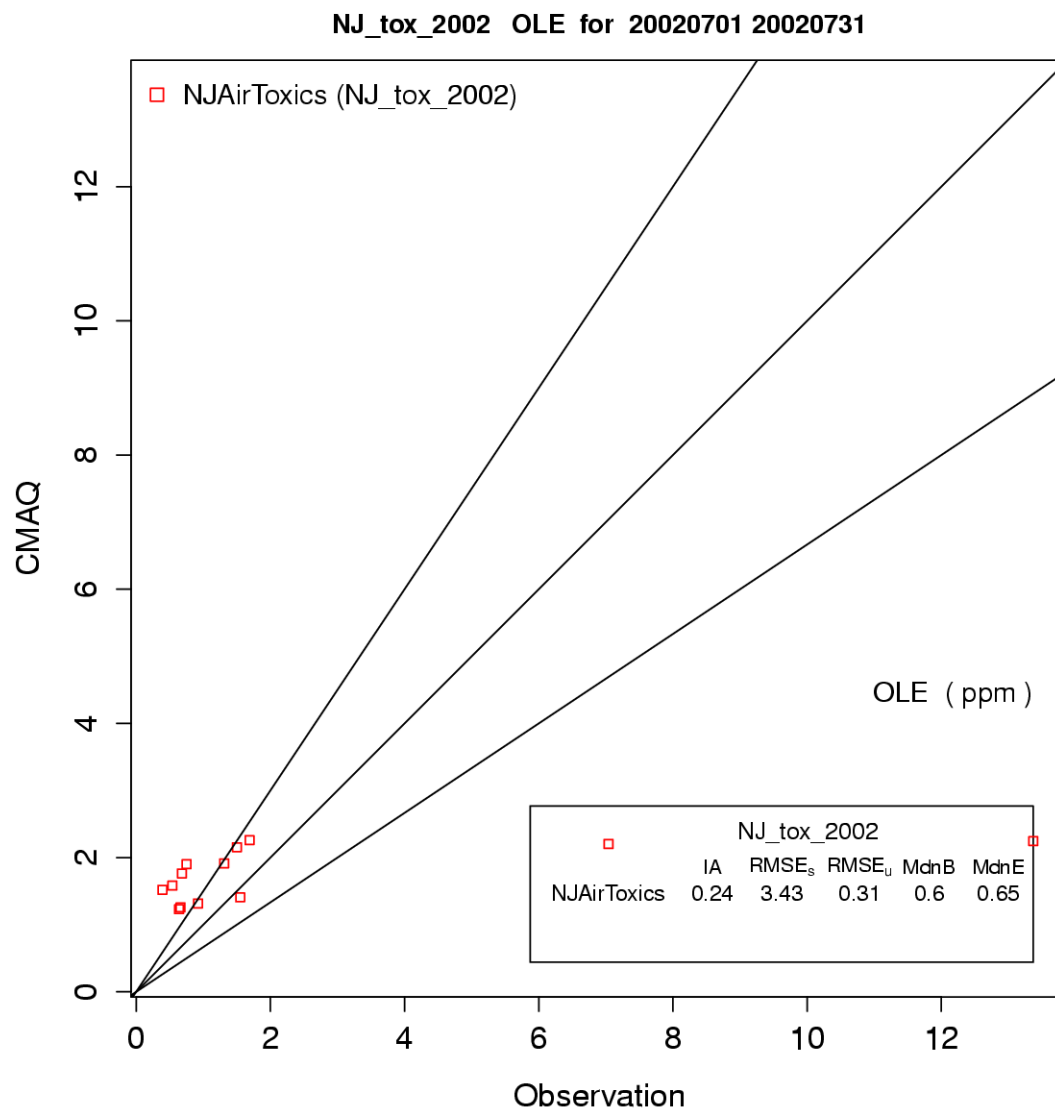


Figure 4-49. Comparison of July scatter plot for paraffins from NJ Air Toxics sites for the air toxics model

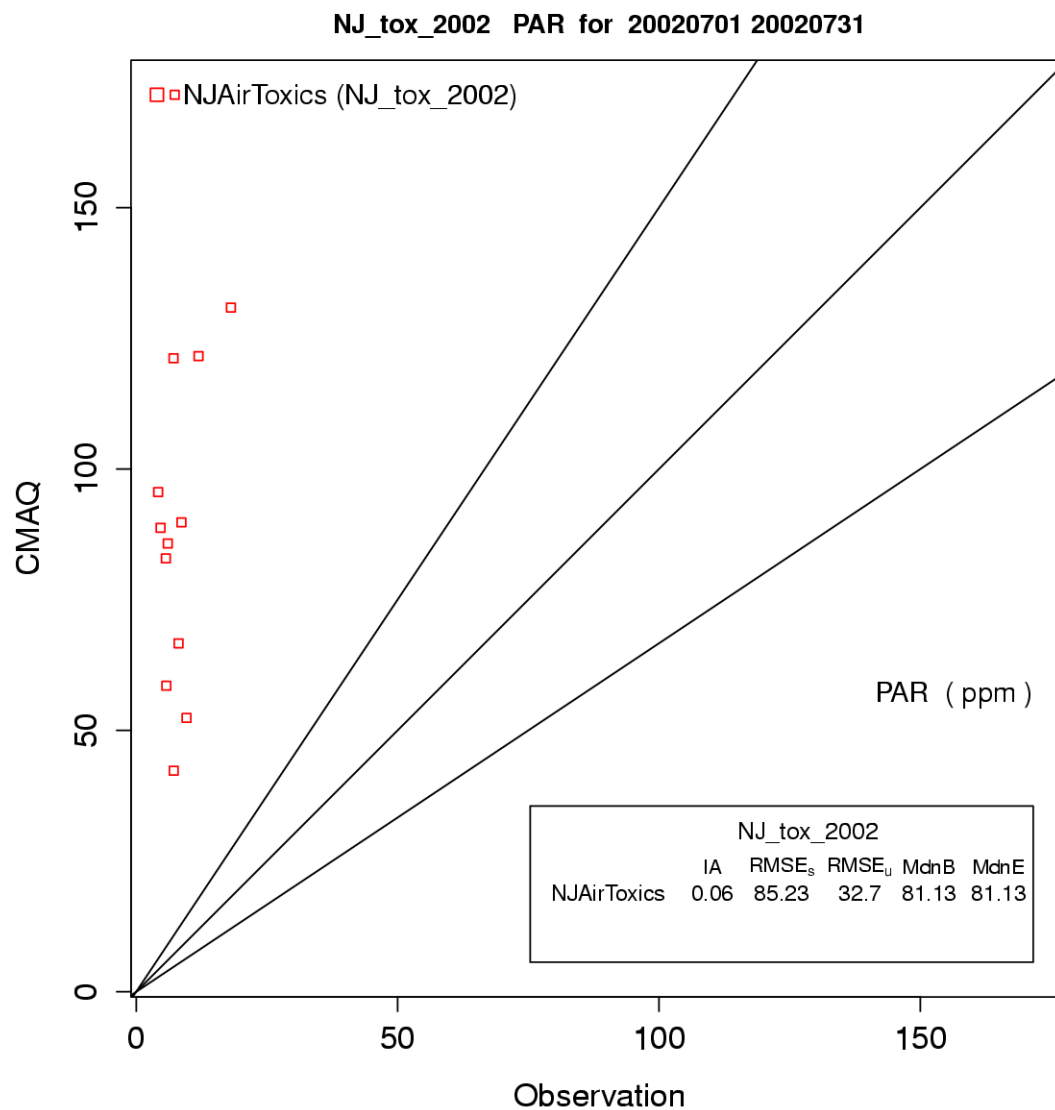


Figure 4-50. Comparison of July scatter plot for toluene from NJ Air Toxics sites for the air toxics model

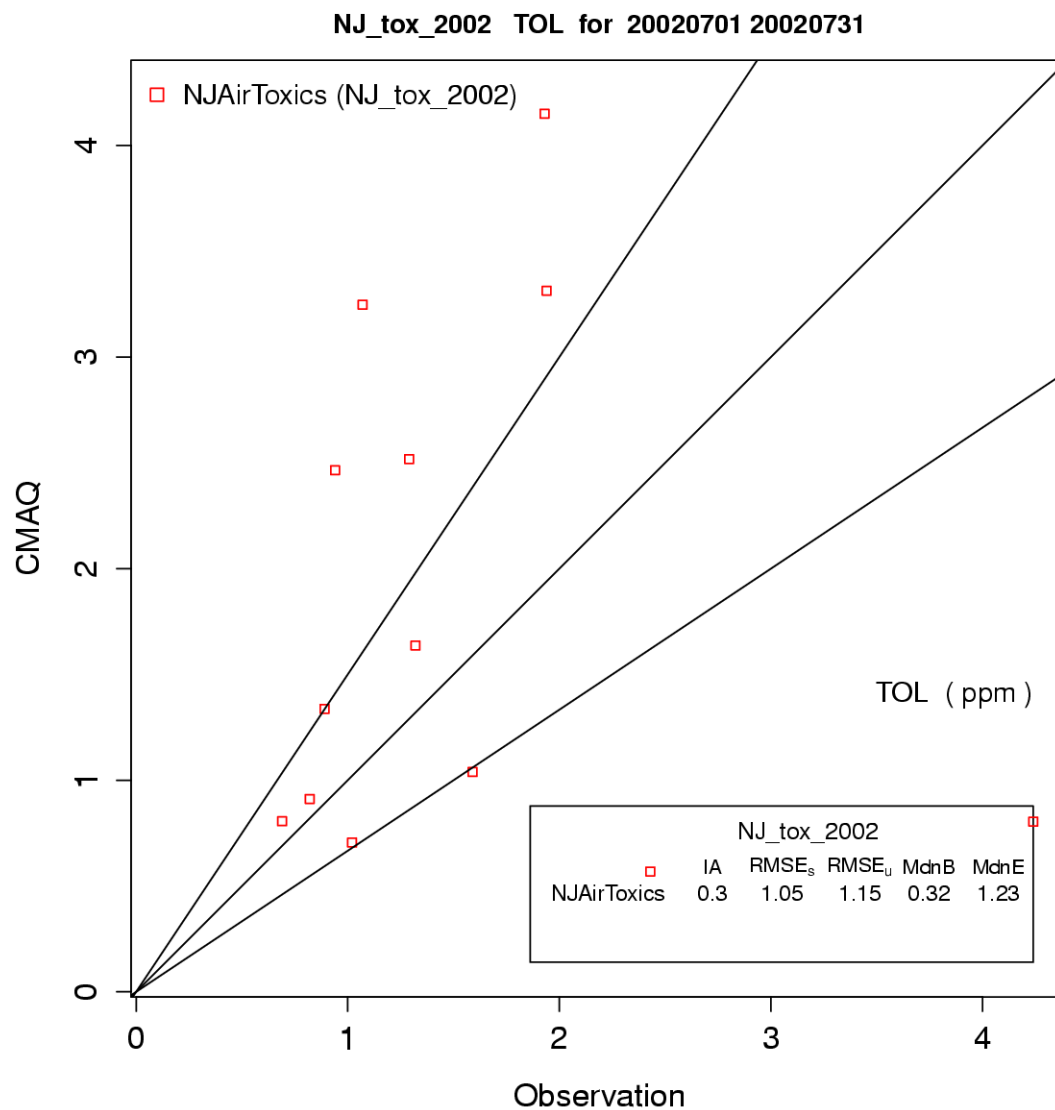
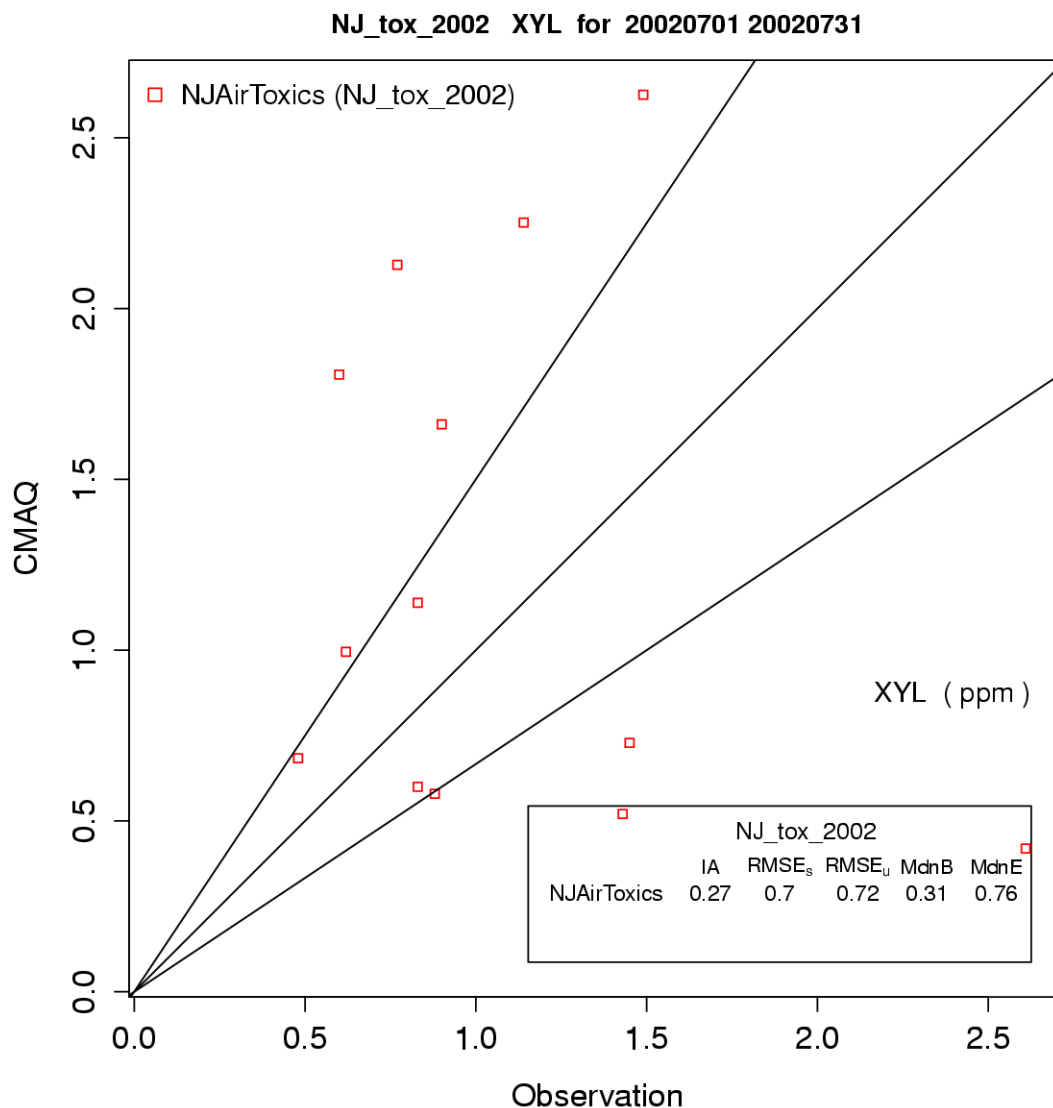


Figure 4-51. Comparison of July scatter plot for xylenes from NJ Air Toxics sites for the air toxics model



4.6. Comparison of the regional haze and air toxics models

Although there are differences in individual aspects of their performance, the regional haze and air toxics models showed similar overall performance. Both were broadly capable of reproducing temporal trends seen statewide, and both had common deficiencies (e.g., the gross overprediction of species with the paraffin C-C bond).

This analysis suggests that large overpredictions seen in the model for CB4 species (which represent aggregates of VOCs, including the air toxics we considered in our previous analysis as described in Section 2) may be due to poor representation of model chemistry or emissions in the model.

4.7. Conclusions about the models

Both models failed to adequately reproduce levels of pollutants observed in New Jersey. These problems in model performance point primarily to the accuracy of the inventory. Because the models were largely able to reproduce the temporal profiles identified in the monitoring data, we conclude that important meteorological processes are relatively well represented in the models. Furthermore, because formaldehyde is reasonably reproduced in both models and it is generated primarily through photochemistry rather than through direct emissions, we conclude that at least some chemical processes are reasonably reproduced in the models. We conclude that the emissions inventory may be grossly overestimating concentrations of air toxics, though additional analysis is required to confirm this result.

Given the relative strengths and weaknesses identified here, we believe that these models are not adequate for assessing absolute levels of air toxics in New Jersey at this time. However, they may be useful in assessing the relative changes expected to result from policy or emission source measures.

5. SUMMARY

This report provides a basis for future analysis of air toxics in New Jersey that can build upon the following key findings:

- The top five risk contributors to cancer risk statewide from NATA 2005 are formaldehyde, benzene, carbon tetrachloride, naphthalene, acetaldehyde, and 1,3-butadiene. The NATA 2005, however, does not assess diesel exhaust for cancer risk in NATA, which NJDEP identifies as the highest cancer risk contributor in New Jersey.⁶
- The top five contributors to respiratory hazard statewide are acrolein, formaldehyde, acetaldehyde, diesel engine emissions, and naphthalene.
- Neurological hazard from ambient air toxics is of low relative importance in New Jersey.
- At a statewide level, the top five contributors to cancer risk, respiratory hazard, and neurological hazard account for 77 percent, 97 percent, and 94 percent of total risk, respectively, according to NATA 2005.
- The US EPA's NATA 2005 model performed reasonably well in reproducing average annual concentrations of key air toxic species.
- Most Group A species had an overall downward trend in annual average concentrations at most New Jersey monitoring sites. Concentrations were typically higher at Elizabeth, followed by Camden, New Brunswick, and Chester.
- Concentrations of carbon tetrachloride are trending higher from year to year. Domestic sources of carbon tetrachloride are dominated by only a few large sources, and by sources in Texas and Louisiana.
- Concentrations of Group C compounds increased or were varied from year to year. Data quality issues raise significant concern over how to interpret these monitoring data.
- Monitoring data for air toxics from the PAMS and NJ Air Toxics networks showed reasonable agreement.
- Data from neighboring states generally exhibit the same temporal trends seen for Group A and C species in New Jersey.
- Both air quality model simulations assessed in this report failed to adequately reproduce levels of air toxics observed in New Jersey.
- Problems in model performance point primarily to the accuracy of the emissions inventory because other processes appear to be adequately represented in the models.

⁶ As previously mentioned, NJDEP and US EPA use different methodologies for determining the health benchmark for diesel exhaust. The results presented here are from US EPA's methodology in NATA 2005.

- While the models assessed in this report are not currently adequate for estimating absolute levels of air toxics, they may be useful in assessing relative changes resulting from policy or emission source measures.

It is important to emphasize that NJDEP interprets the data from NATA differently than does the US EPA, because NJDEP uses different health benchmarks. Of special note, while the US EPA did not assess cancer risk from diesel exhaust, NJDEP uses the California cancer risk factor to determine the health benchmark for diesel particulate matter. Under the NJDEP approach, diesel exhaust is the highest contributor for cancer risk in the state, and would have been the greatest contributor to cancer risk in the 2005 NATA assessment had it been included. NJDEP estimates that cancer risk from exposure to diesel exhaust averaged across the state is 324 in a million, which is an order of magnitude higher than that of formaldehyde, the compound having the highest ranked cancer risk in NATA 2005.

6. REFERENCES

- Allen, N.D.C., P.F. Bernath, C.D. Boone, M.P. Chipperfield, D. Fu, G.L. Manney, D.E. Oram, G.C. Toon, and D.K. Weisenstein. (2009). Global carbon tetrachloride distributions obtained from the Atmospheric Chemistry Experiment (ACE). *Atmospheric Chemistry and Physics*, 9:7449-7459.
- Appel, K.W., A.B. Gilliland, G. Sarwar, and R.C. Gilliam. (2007). Evaluation of the Community Multiscale Air Quality (CMAQ) model version 4.5: Sensitivities impacting model performance Part I-ozone. *Atmospheric Environment* 41(40):9603-9615.
- Appel, K.W., P.V. Bhawe, A.B. Gilliland, G. Sarwar, and S.J. Roselle. (2008). Evaluation of the community multiscale air quality (CMAQ) model version 4.5: Sensitivities impacting model performance; Part II—particulate matter. *Atmospheric Environment* 42(24):6057-6066.
- Appel, K.W., S.J. Roselle, R.C. Gilliam, and J.E. Pleim. (2009). Sensitivity of the Community Multiscale Air Quality (CMAQ) model v 4.7 results for the eastern United States to MM5 and WRF meteorological drivers. *Geosci. Model Dev. Discuss.* 2:1081-1114.
- ATSDR (Agency for Toxic Substances and Disease Registry). (2005). Division of Toxicology ToxFAQ for Carbon Tetrachloride. Available at: <http://www.atsdr.cdc.gov/tfacts30.pdf>
- ATSDR (Agency for Toxic Substances and Disease Registry). (2006). Division of Toxicology ToxFAQ for Cyanide. Available at: <http://www.atsdr.cdc.gov/tfacts8.pdf>
- ATSDR (Agency for Toxic Substances and Disease Registry). (2007). Medical Management Guidelines for Methyl Bromide. Available at: <http://www.atsdr.cdc.gov/mHmI/mmg27.pdf>
- Bortnick, S.M. and S.L. Stetzer. (2002). Sources of variability in ambient air toxics monitoring data. *Atmospheric Environment* 36, 1783-1791.
- Boylan, J.W. and A.G. Russell. (2006). PM and light extinction model performance metrics, goals, and criteria for three-dimensional air quality models. *Atmospheric Environment*, 40(26):4946-4959.
- CARB (California Air Resources Board). (2005). Summary of Adverse Impacts of Diesel Particulate Matter. Available at: http://www.cleanairco.com/public/pdf/diesel_health_effects_summary_7-5-05-1.pdf
- CARB (California Air Resources Board). (2011). Diesel & Health Research. Available at <http://www.arb.ca.gov/research/diesel/diesel-health.htm>
- Carter, W.P.L. (2011). Development of an Improved Chemical Speciation Database for Processing Emissions of Volatile Organic Compounds for Air Quality Models. Accessed: September 19, 2011. Available at: <http://www.engr.ucr.edu/~carter/emitdb/>

- Ching, J. S., T.E. Pierce Jr., T. Palma, W.T. Hutzell, R. Tang, A. Cimorelli, and J. Herwehe. (2004). Linking air toxic concentrations from CMAQ to the HAPEM5 exposure model at neighborhood scales for the Philadelphia area. Presented at 5th Symposium on the Urban Environment, Vancouver, BC, Canada, August 23-27, 2004.
- Ching, J., J. Herwehe, and J. Swall. (2006a). On joint deterministic grid modeling and sub-grid variability conceptual framework for model evaluation. *Atmospheric Environment*, 40(26):4935-4945.
- Ching, J., V. Isakov, M.A. Majeed, and J.S. Irwin. (2006b). An approach for incorporating sub-grid variability information into air quality modeling. Paper 2.1. 14th Joint Conference on the Applications of Air Pollution Meteorology with the Air and Waste Management Association.
- Eastern Research Group, Inc. (2008). 2007 National Monitoring Programs (UATMP and NATTS) Volume I: Main Content. Final report for the US EPA. EPA-454/R-08-008a
- Gelfand, A.E., L. Zhu, and B.P. Carlin. (2000). On the change of support problem for spatio-temporal data. *Biostatistics* 2(1):31-45.
- Gery, M.W., G.Z. Whitten, J.P. Killus, and M.C. Dodge. 1989. A Photochemical Kinetics Mechanism for Urban and Regional Scale Computer Modeling. *J. Geophys. Res.*, 94:925-956.
Note: The CB4 chemical mechanism has been updated by others in the 1990s. See Yarwood et al. (2005) for a more detailed history of the mechanism.
- Gilliam, R, W. Appel, and S. Phillips. (2005). The Atmospheric Model Evaluation Tool: Meteorology Module. Presented at the 4th Annual CMAS Conference, Chapel Hill, NC, September 2005. Also available at:
http://www.cmascenter.org/conference/2005/abstracts/6_1.pdf
- Hafner, H.R., M.C. McCarthy, N.J.M. Wheeler, and M. Koerber. (2004). Policy-Relevant Lessons Learned from Phase III Air Toxics Analyses: White Paper. Petaluma (CA): Sonoma Technology, Inc. (STI). August 10, 2004. STI-903553-2585-WP. Prepared for Lake Michigan Air Directors Consortium (LADCO).
- Hopke, P.K. (2000). A Guide to Positive Matrix Factorization, EPA Workshop Proceedings, Materials from the Workshop on UNMIX and PMF as Applied to PM_{2.5}, February 14-16, 2000.
- Hopke, P.K. (2002). Initial Investigation of PAMS Data Using Positive Matrix Factorization (PMF). Report to Sonoma Technology, Inc.
- ICF International. (2011). An Overview of Methods for EPA's National-Scale Air Toxics Assessment. Research Triangle Park, N.C.: U.S. Environmental Protection Agency (EPA), Office of Air Quality, Planning, and Standards (OAQPS). January 2011.
- Isakov, V. and A. Venkatram. (2006). Resolving neighborhood scale in air toxics modeling: a case study in Wilmington, CA. *J. Air & Waste. Manage. Assoc.* 56:559-568.

- Isakov, V., J.S. Irwin, and J. Ching. (2007). Using CMAQ for exposure modeling and characterizing the subgrid variability for exposure estimates. *Journal of Applied Meteorology and Climatology*, 46:1354-1371.
- Jones, M. (2010). Data quality evaluation guidelines for ambient air acrolein measurements. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, December 17, 2010. Available at: <http://www.epa.gov/ttnamti1/files/ambient/airtox/20101217acroleindataqualityeval.pdf> (accessed June 7, 2011).
- Karamchandani, P., K. Lohman, and C. Seigneur. (2009). Using a sub-grid scale modeling approach to simulate the transport and fate of toxic air pollutants. *Environ Fluid Mech.* 9:59–71.
- Luecken, D.J., W.T. Hutzell, and G.L. Gipson. (2006). Development and analysis of air quality modeling simulations for hazardous air pollutants. *Atmospheric Environment*, 40(26):5087-5096.
- Martinez, M., H. Harder, X. Ren, R.L. Lesher, and W.H. Brune. (2004). Measuring atmospheric naphthalene with laser-induced fluorescence. *Atmospheric Chemistry and Physics Discussions*, 4(1):343–363.
- McCarthy, M.C., H.R. Hafner, L.R. Chinkin, and J. G. Charrier. (2007). Temporal variability of selected air toxics in the United States. *Atmospheric Environment* 41(34):7180-7194. doi:10.1016/j.atmosenv.2007.05.037
- McNair, L.A., R.A. Harley, and A.G. Russell. (1996). Spatial inhomogeneity in pollutant concentrations and their implications for air quality model evaluation. *Atmospheric Environment* 30(24):4291-4301.
- NESCAUM (Northeast States for Coordinated Air Use Management). (2006). Contributions to Regional Haze in the Northeast and Mid-Atlantic United States, Mid-Atlantic/Northeast Visibility Union (MANE-VU) Contribution Assessment, Appendix C: Chemical Transport Model Results for Sulfate Source Attribution Studies in the Northeast U.S., Model description and performance evaluation. NESCAUM, Boston, MA. Available at: <http://www.nescaum.org/documents/contributions-to-regional-haze-in-the-northeast-and-mid-atlantic--united-states/>
- NJDEP (New Jersey Department of Environmental Protection). (2003). 2003 Photochemical Assessment Monitoring Stations (PAMS). Available at: <http://www.state.nj.us/dep/airmon/pams03.pdf>
- NJDEP (New Jersey Department of Environmental Protection). (2011). Air Toxics in New Jersey: Diesel Emissions. Available at <http://www.nj.gov/dep/airtoxics/diesemis.htm>
- OEHHA (California Office of Environmental Health Hazard Assessment). 2000. Chronic Toxicity Summary of Phthalic Anhydride. Available at: http://oehha.ca.gov/air/chronic_rels/pdf/85449.pdf

- Park S., C.E. Cobb, K. Wade, J. Mulholland, Y. Hu, and A.G. Russell. (2006). Uncertainty in air quality model evaluation for particulate matter due to spatial variations in pollutant concentrations. *Atmospheric Environment*, 40:563-573.
- Pietarinen, C. (2011). E-mail exchange with NESCAUM. June 7, 2011.
- Seinfeld, J. and S. Pandis. (1998). *Atmospheric Chemistry and Physics: From Air Pollution to Climate Change*. John Wiley and Sons, Inc., New York, pp. 89,109, 111, 177, 244, 281-2, 288, 312, 378, 733-4.
- Sistla, G. and N. Aleksic. (2007). A comparison of PAMS and air toxics measurements. *Atmospheric Environment*, 41:5719-5731.
- Seigneur, C., B. Pun, K. Lohman, and S.-Y. Wu. (2003). Regional Modeling of the Atmospheric Fate and Transport of Benzene and Diesel Particles. *Environ. Sci. Technol.*, 37(22):5236–5246.
- Spicer, C.W., B.E. Buxton, M.W. Holdren, D.L. Smith, T.J. Kelly, S.W. Rust, A.D. Pate, G.M. Sverdrup, and J.C. Chuang. (1996). Variability of hazardous air pollutants in an urban area. *Atmospheric Environment* 30(20):3443-3456.
- Stein, A.F., V. Isakov, J. Godowitch, and R.R. Draxler. (2007). A hybrid modeling approach to resolve pollutant concentrations in an urban area. *Atmospheric Environment*, 41:9410-9426.
- Touma, J.S., V. Isakov, J. Ching, and C. Seigneur. (2006). Air quality modeling of hazardous pollutants: current status and future directions. *J. Air & Waste Manage. Assoc.* 56:547-558.
- US EPA (U.S. Environmental Protection Agency). (1994). Environmental Fact Sheet: Air Toxics from Motor Vehicles. EPA400-F-92-004. Available at: <http://www.epa.gov/oms/f02004.pdf>
- US EPA (U.S. Environmental Protection Agency). (2002). Health Assessment Document for Diesel Engine Exhaust. Prepared by the National Center for Environmental Assessment, Washington, DC, for the Office of Transportation and Air Quality; EPA/600/8-90/057F.
- US EPA (U.S. Environmental Protection Agency). (2006). 2006-2011 EPA Strategic Plan: Charting Our Course. Washington, DC: US EPA. September 2006.
- US EPA (U.S. Environmental Protection Agency). (2007). Guidance on the Use of Models and Other Analyses for Demonstrating Attainment of Air Quality Goals for Ozone, PM_{2.5}, and Regional Haze. EPA -454/B-07-002.
- US EPA (U.S. Environmental Protection Agency). (2008) 2005 National Emissions Inventory. "ALLNEI HAP Annual 01233008" database. Available at: <http://www.epa.gov/ttnchie1/net/2005inventory.html>.
- US EPA (U.S. Environmental Protection Agency). (2009a) National-Scale Air Toxics Assessment for 2002 - Fact Sheet. Available at: <http://www.epa.gov/ttn/atw/nata2002/factsheet.html>

- US EPA (U.S. Environmental Protection Agency). (2009b). Health Effects Notebook for Hazardous Air Pollutants. Available at:
<http://www.epa.gov/ttn/atw/hlthef/hapindex.html>
- US EPA (U.S. Environmental Protection Agency). (2010a). Data Quality Evaluation Guidelines for Ambient Air Acrolein Measurements. Last updated: December 17, 2010. Accessed on: Jun 7, 2011. Available at:
<http://www.epa.gov/ttnamti1/files/ambient/airtox/20101217acroleindataqualityeval.pdf>.
- US EPA (U.S. Environmental Protection Agency). (2010b). EPA Schools Monitoring Initiative Fact Sheet, March 2010 Update: Acrolein Monitoring Results Are Uncertain. Available at: <http://www.epa.gov/schoolair/pdfs/acroleinupdate.pdf>.
- US EPA (U.S. Environmental Protection Agency). (2011). National-Scale Air Toxics Assessment for 2005, Fact Sheet.
- US EPA (U.S. Environmental Protection Agency). (2012). Integrated Risk Information System (IRIS): Diesel engine exhaust. Available at:
<http://www.epa.gov/iris/subst/0642.htm>
- Yarwood, G., S. Rao, M. Yocke, and G. Z. Whitten. (2005). Updates to the carbon bond chemical mechanism: CB05. Final report to the US EPA, RT-0400675. Available online at http://www.camx.com/Files/CB05_Final_Report_120805.aspx
- Yu, S., B. Eder, R. Dennis, S. Chu, and S.E. Schwartz. (2006). New unbiased symmetric metrics for evaluation of air quality models. *Atmospheric Science Letters* 7:26-34.
- Zhu, X., Z. Fan, X. Wu, Q. Meng, S. Wang, S., X. Tang, P. Ohman-Strickland, P. Georgopoulos, J. Zhang, L. Bonanno, H. Held, and P. Liroy. (2008). Spatial variation of volatile organic compounds in a “hot spot” for air pollution. *Atmospheric Environment* 42 (32), 7329-7338.

Appendix A: Comparison of photochemical assessment monitoring stations and New Jersey air toxics site data

Appendix A: Comparison of photochemical assessment monitoring stations and New Jersey air toxics site data

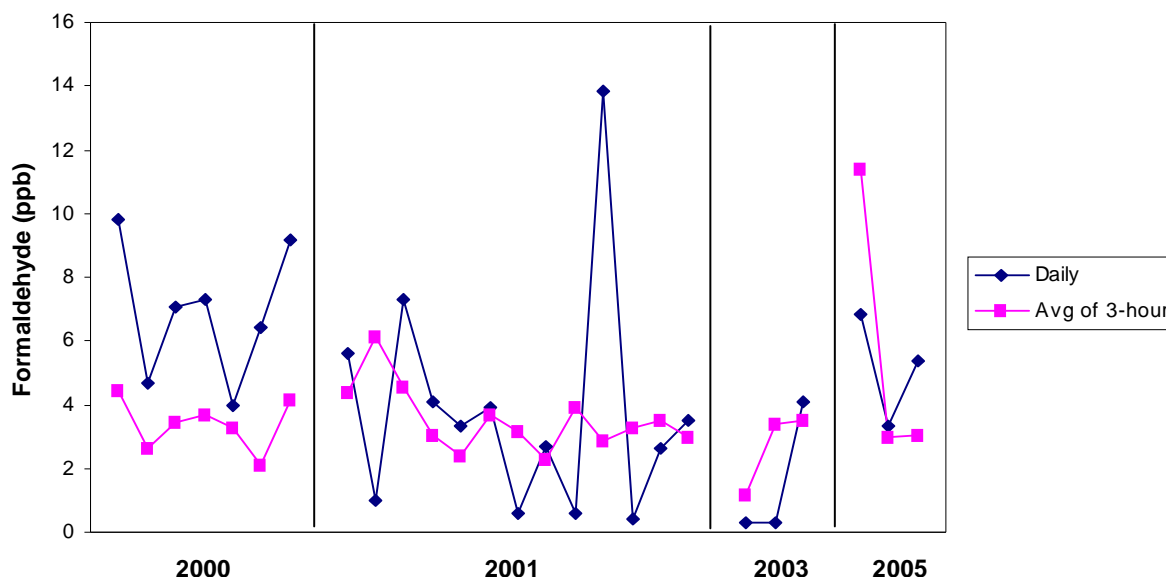
While 24-hour samples are typically collected at air toxics measurement sites (NJ Air Toxics), hourly (or 3-hour average, in the case of carbonyls) samples are collected at Photochemical Assessment Monitoring Stations (PAMS) during the summer ozone season. PAMS sites measure a host of VOCs, and a subset of those species are also measured as part of the toxics program. In Section 3.4.4, the PAMS toxics measurements were used to get a sense for the diurnal behavior of those toxics species measured every 24 hours at the NJ Air Toxics measurement sites.

In this section, 24-hour NJ Air Toxics measurements and averaged hourly PAMS measurements are compared for collocated monitors (at Camden) and concurrent measurement days.

Formaldehyde and acetaldehyde

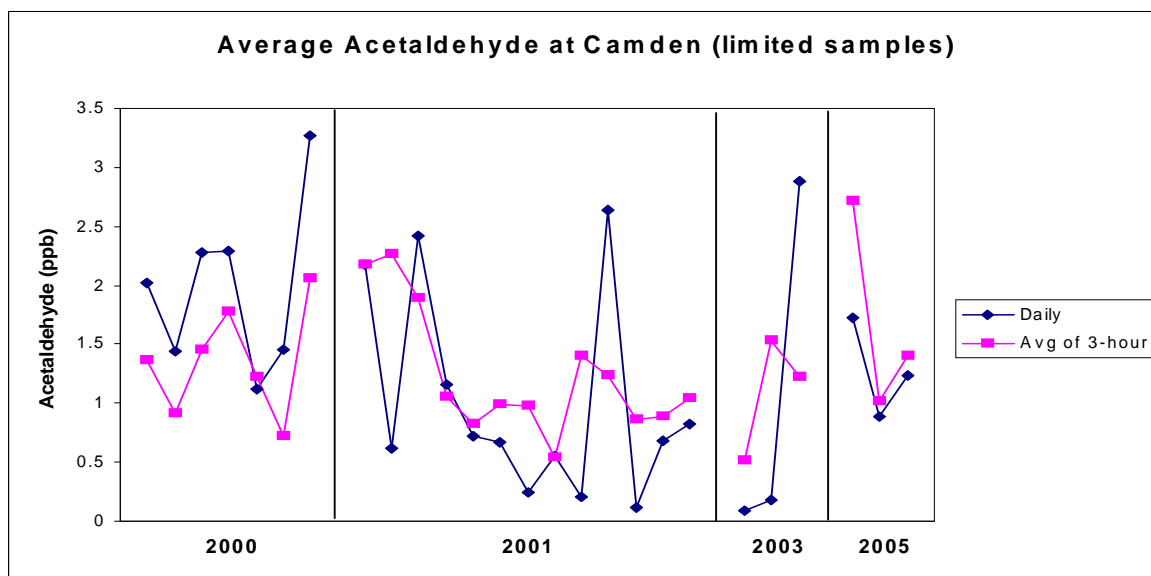
Three-hour PAMS measurements of formaldehyde and acetaldehyde were only made during a subset of the years examined here. These measurements were averaged to generate 24-hour averaged PAMS data for comparison with the 24-hour NJ Air Toxics measurements. Data for the limited number of days for which measurements were made at both collocated samplers (at Camden) are given in Figure A-1 for formaldehyde and Figure A-2 for acetaldehyde. While the points are evenly spaced along the x-axis, the days for which there are matching data are not necessarily evenly spaced. Note that if there was more than one entry for a NJ Air Toxics measurement on a given day, the entries were averaged. In 2000, for the five matching data points, the trend was similar between PAMS and NJ Air Toxics samplers for both formaldehyde and acetaldehyde, but PAMS measurements (pink line with squares) were always lower than NJ Air Toxics measurements (blue line with diamonds). In 2001, the PAMS and NJ Air Toxics concentrations were similar, but there were a few days when one monitor failed to capture the peak of another. There were only three points each in 2003 and 2005 for comparison. There were significant differences for some days, but the overall concentration levels were similar for both sampler types. These differences underscore the uncertainty around data quality for these compounds discussed in Section 3.4.5.

Figure A-1. Comparison of averaged PAMS and NJ Air Toxics measurements for formaldehyde at Camden



Note: Measurements were collected on the same days for collocated monitors. Only days with 75 percent data completeness (at least 18 hours) were included. Daily measurements are from the NJ Air Toxics site; three-hour data from the PAMS network are averaged over the 24-hour period to match with the NJ Air Toxics data. Note that all days that passed the 75 percent data completeness requirement were actually 100 percent complete.

Figure A-2. Comparison of averaged PAMS and NJ Air Toxics measurements for acetaldehyde at Camden

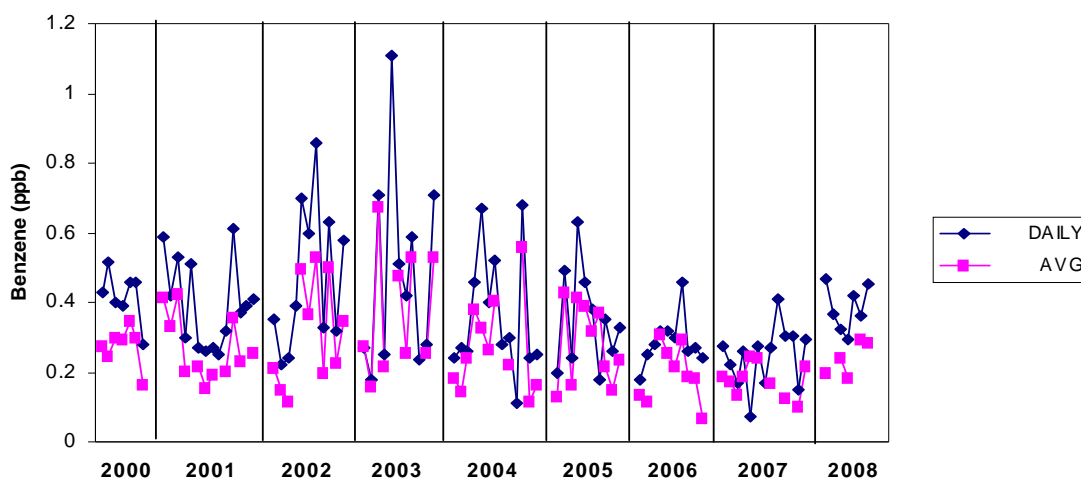


Note: Measurements were collected on the same days for collocated monitors. Only days with 75 percent data completeness (at least 18 hours) were included. Daily measurements are from the NJ Air Toxics site; three-hour data from the PAMS network are averaged over the 24-hour period to match with the NJ Air Toxics data. Note that all days that passed the 75 percent data completeness requirement were actually 100 percent complete.

Benzene

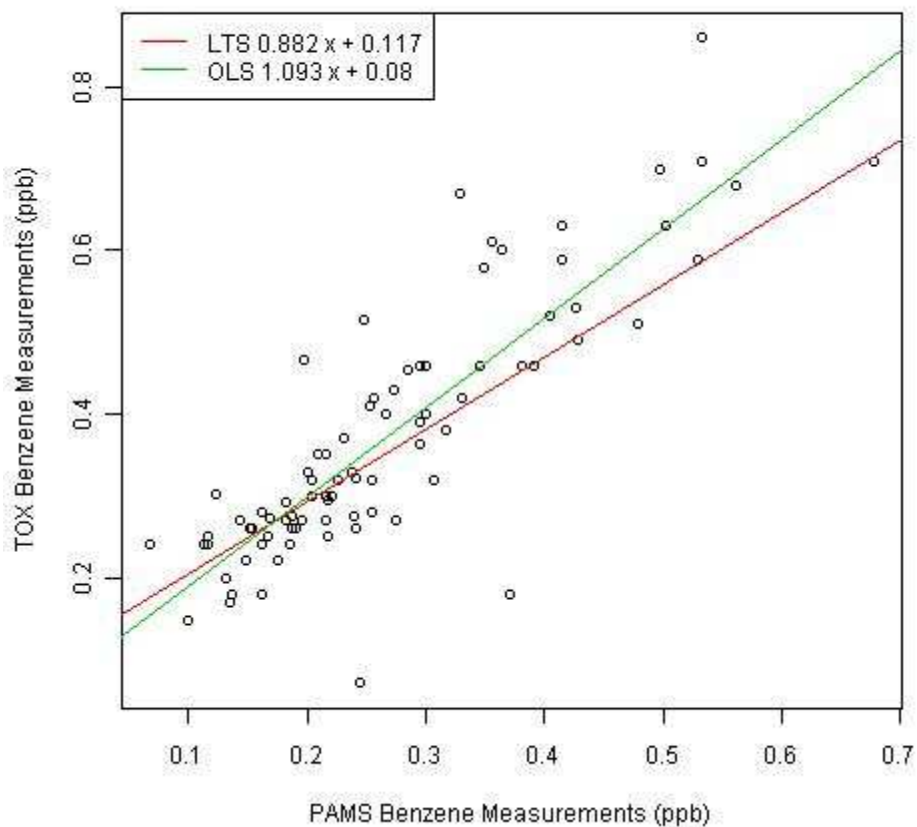
Figure A-3 and Figure A-4 show time-series and scatter plots of benzene measurements for the 24-hour air toxics (NJ Air Toxics marked as TOX, as a blue line with diamonds) and PAMS (pink line with squares) samplers. PAMS measurements were averaged to generate data for comparison with the 24-hour measurements. There were only limited data during each year for which there were simultaneous PAMS and NJ Air Toxics measurements. For most years in Figure A-3, there was good agreement between the PAMS and NJ Air Toxics benzene data. In Figure A-4, in addition to the observational data, the results of two different methods of linear regression are included – 1) ordinary least squares (OLS), the method that is typically used in Excel charts and is best for data without large outliers, and 2) least trimmed squares (LST), which is considered a robust regression method that is less sensitive to outliers. In a comparison of PAMS and NJ Air Toxics data at sites in New York, Sistla and Aleksic (2007) used least trimmed squares to assess the linear relationship between PAMS and NJ Air Toxics data, because it was robust and “essentially unaffected by extreme values.” For both regressions, the intercepts are not very high and estimated slopes are not very far from one, indicating that PAMS and NJ Air Toxics daily concentrations for benzene are similar for the limited data comparison. Summer averages for both datasets also indicate that both datasets are comparable for most years (Figure A-5).

Figure A-3. Comparison of averaged PAMS and NJ Air Toxics measurements for benzene at Camden



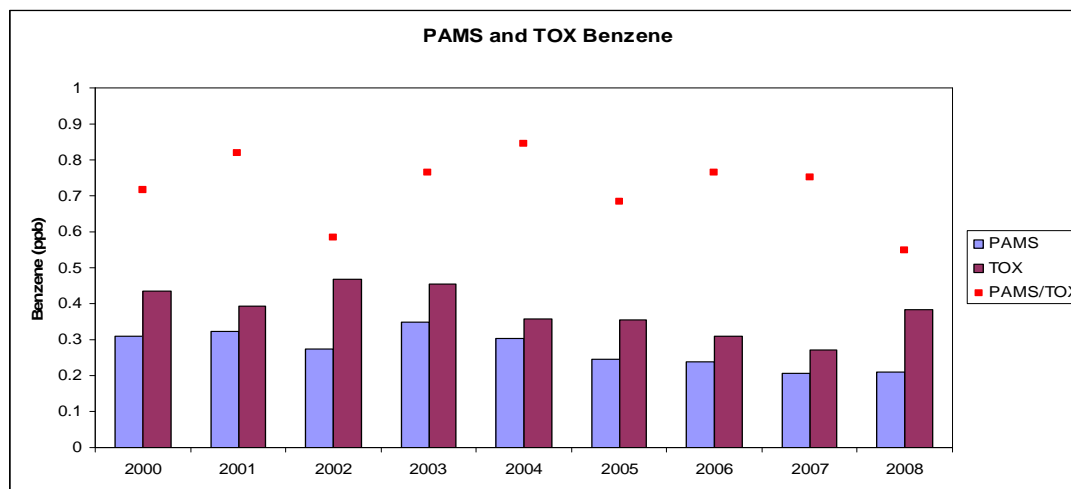
Note: Measurements were collected on the same days for collocated monitors. Only days with 75 percent data completeness (at least 18 hours) were included. Daily measurements are from the NJ Air Toxics site; hourly data from the PAMS network are averaged over the 24-hour period to match with the NJ Air Toxics data.

Figure A-4. Scatter plot of averaged PAMS and NJ Air Toxics measurements for benzene at Camden



Note: Measurements were collected on the same days for collocated monitors. Only days with 75 percent data completeness (at least 18 hours) were included. Daily measurements are from the NJ Air Toxics site; hourly data from the PAMS network are averaged over the 24-hour period to match with the NJ Air Toxics data. Two different methods of linear regression were used to develop trend lines, ordinary least squares (OLS) and least trimmed squares (LTS).

Figure A-5. Summer average PAMS and NJ Air Toxics measurements for benzene at Camden for 2000-2008

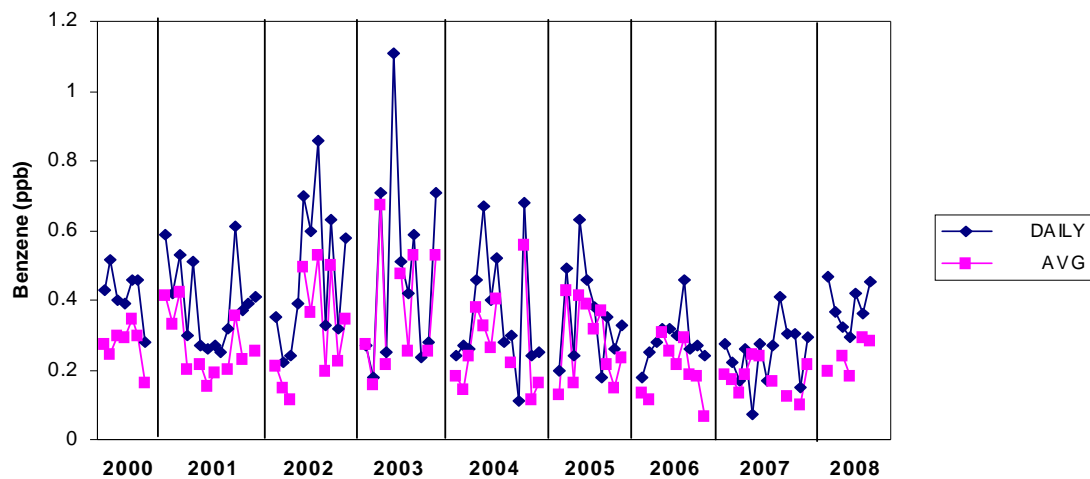


Note: Half of the minimum detection limit (MDL) was substituted for data points below the MDL, but no data completeness restrictions were imposed on the data used in the averages.

Xylenes

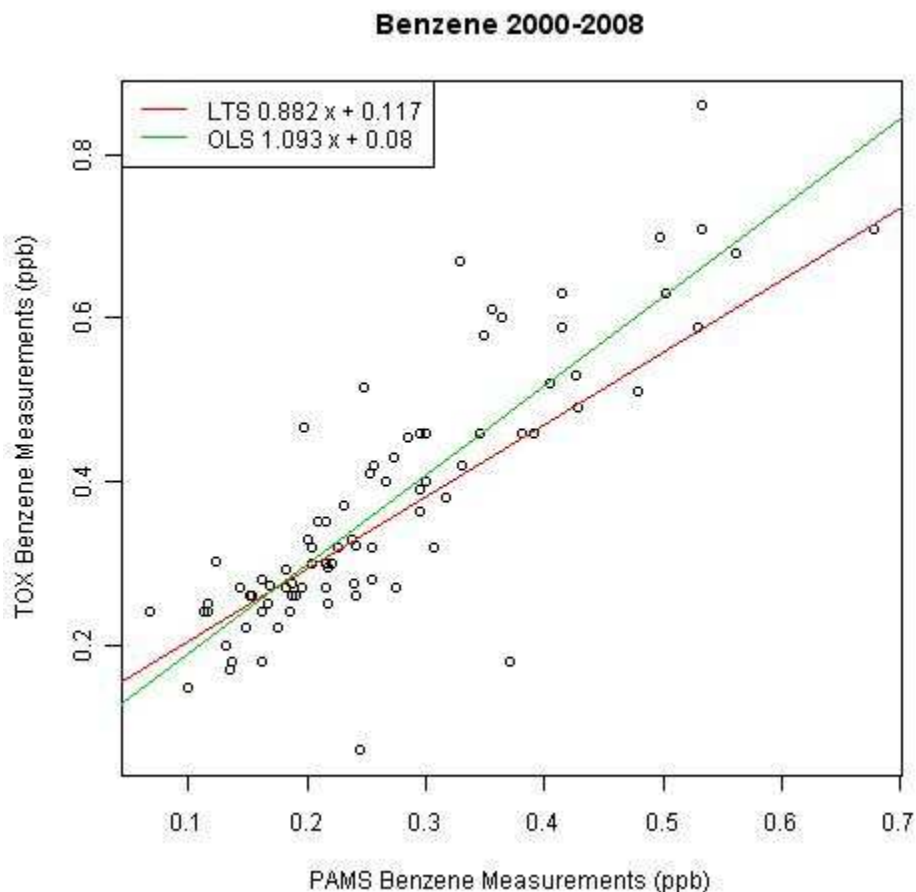
Hourly PAMS measurements of *m,p*-xylenes and *o*-xylene isomers were available for 2000-2008. These measurements were averaged to generate 24-hour averaged PAMS data for comparison with the 24-hour NJ Air Toxics measurements on days when both measurements were taken and PAMS data were at least 75 percent complete. The *m,p*-xylenes and *o*-xylenes were combined for comparison. Figure A-6 contains a time-series plot of the two datasets, the average of the hourly PAMS data (pink line with squares) and the daily NJ Air Toxics measurements (blue line with diamonds). A scatter plot of NJ Air Toxics and PAMS xylenes data is given in Figure A-7. Figure A-7 also includes the results of two different methods of linear regression. During some years in Figure A-6 there was good agreement between the PAMS and NJ Air Toxics xylenes data (e.g., 2003-2004). There were other years, when there were substantial deviations (e.g., 2000). On average, the daily NJ Air Toxics xylenes concentrations are higher than the averaged hourly PAMS concentrations. There is a significant amount of scatter in the points in Figure A-7, and OLS and LTS regressions give slopes of 0.7 and 0.6, respectively. This indicates more substantial differences in the daily average xylenes concentrations measured by the two collocated monitors compared to benzene. Summer averages of *m,p*-xylenes and *o*-xylene for both datasets indicate, however, that both datasets are comparable for most years (Figure A-8).

Figure A-6. Comparison of averaged PAMS and NJ Air Toxics measurements for xylenes at Camden



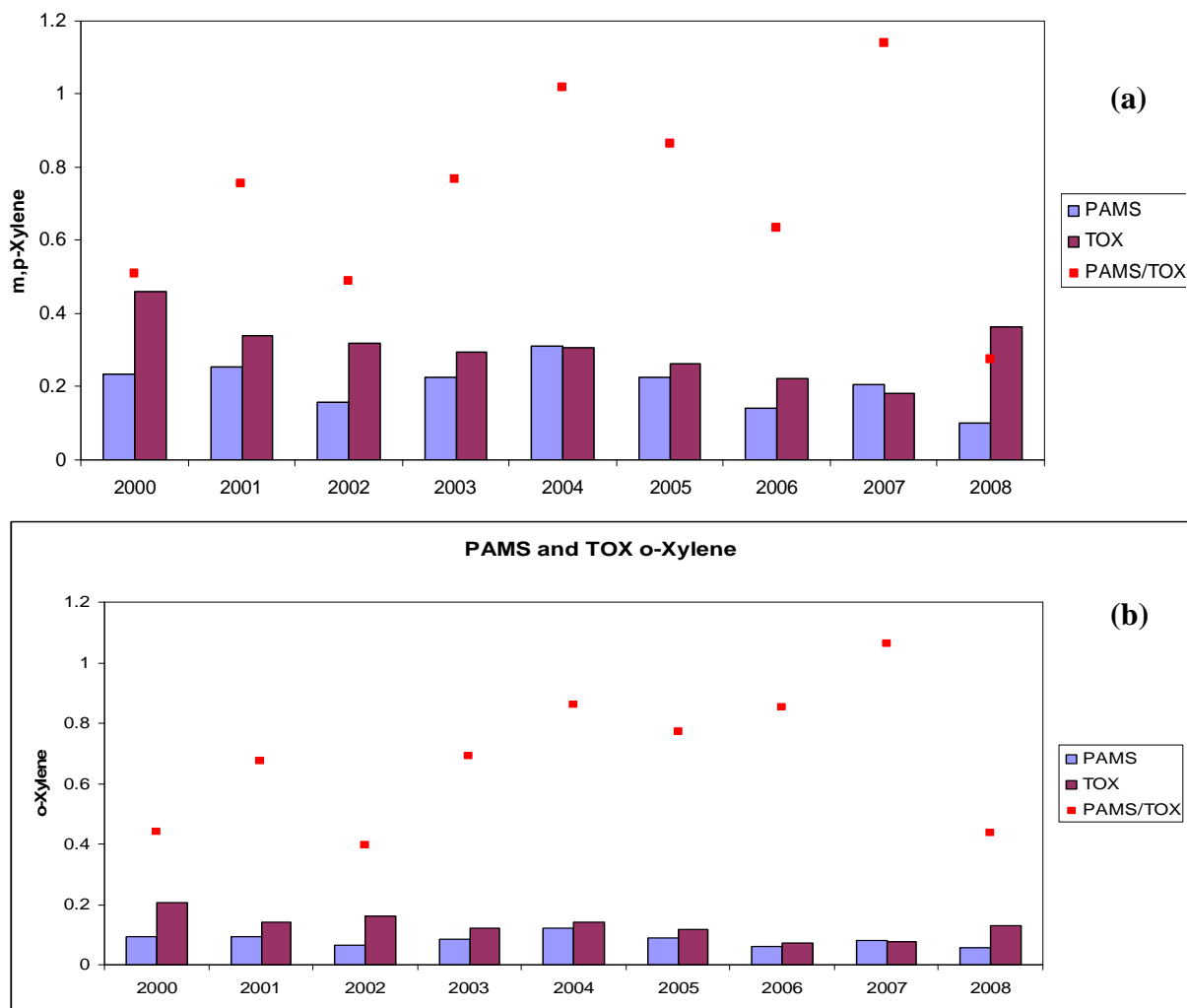
Note: Measurements were collected on the same days for collocated monitors. Only days with 75 percent data completeness (at least 18 hours) were included. Daily measurements are from the NJ Air Toxics site; hourly data from the PAMS network are averaged over the 24-hour period to match with the NJ Air Toxics data.

Figure A-7. Scatter plot of averaged PAMS and NJ Air Toxics measurements for xylenes at Camden



Note: Measurements were collected on the same days for collocated monitors. Only days with 75 percent data completeness (at least 18 hours) were included. Daily measurements are from the NJ Air Toxics site; hourly data from the PAMS network are averaged over the 24-hour period to match with the NJ Air Toxics data. Two different methods of linear regression were used to develop trend lines, ordinary least squares (OLS) and least trimmed squares (LTS).

Figure A-8. Summer average PAMS and NJ Air Toxics measurements for xylenes at Camden for 2000-2008



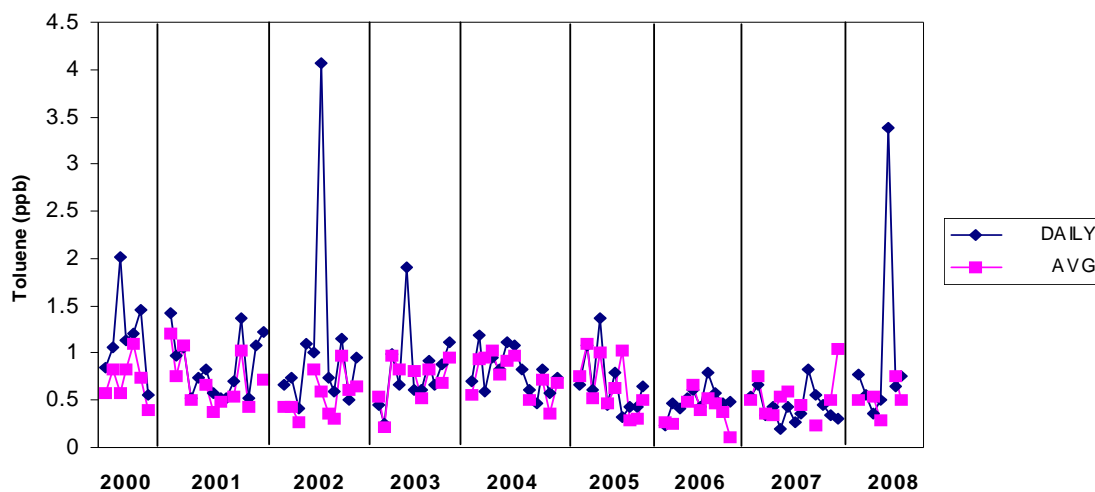
Note: Half of the minimum detection limit (MDL) was substituted for data points below the MDL, but no data completeness restrictions were imposed on the data used in the averages.

(a) *m,p*-Xylenes. (b) *o*-Xylene.

Toluene

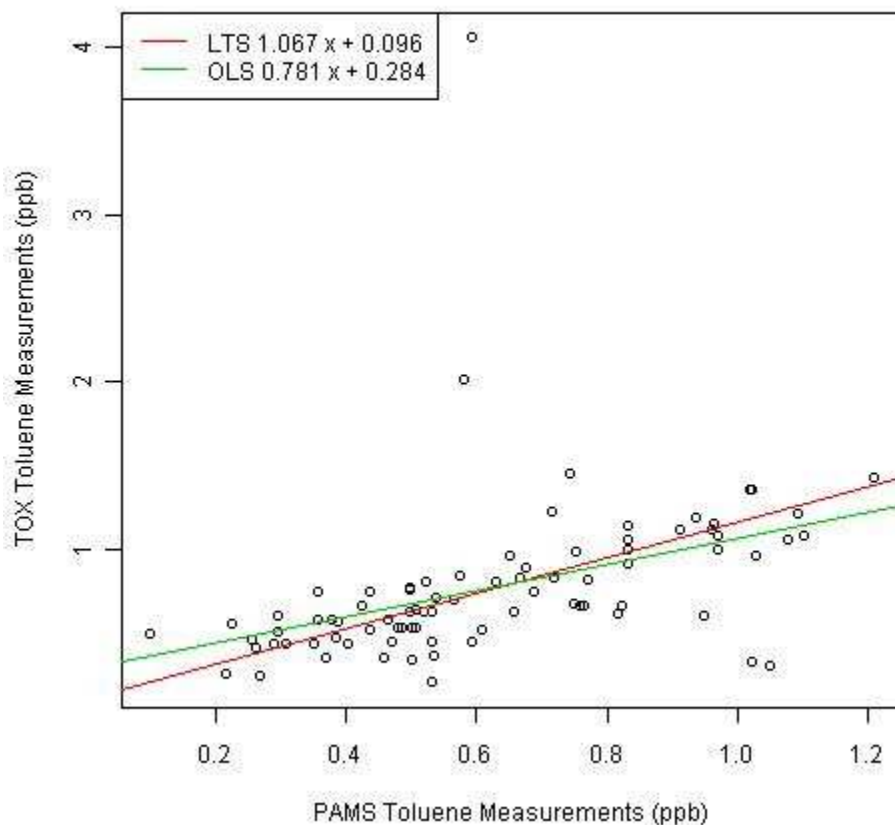
Figure A-9 and Figure A-10 show time-series and scatter plots of toluene measurements for the NJ Air Toxics (blue line with diamonds) and PAMS (pink line with squares) samplers. PAMS and NJ Air Toxics toluene concentrations are very similar for the most part, though there are several peaks in the NJ Air Toxics data that are not seen in the averaged PAMS data for that same day. The scatter plot for toluene (Figure A-10) shows an obvious linear relationship between concentrations given by the two monitors, though there are a number of “outliers.” A least trimmed squares regression has a low intercept and a slope close to one, indicating that the PAMS and NJ Air Toxics daily measurements for toluene are very similar. Summer averages of toluene for both datasets also indicate that both datasets are comparable for most years (Figure A-11).

Figure A-9. Comparison of averaged PAMS and NJ Air Toxics measurements for toluene at Camden



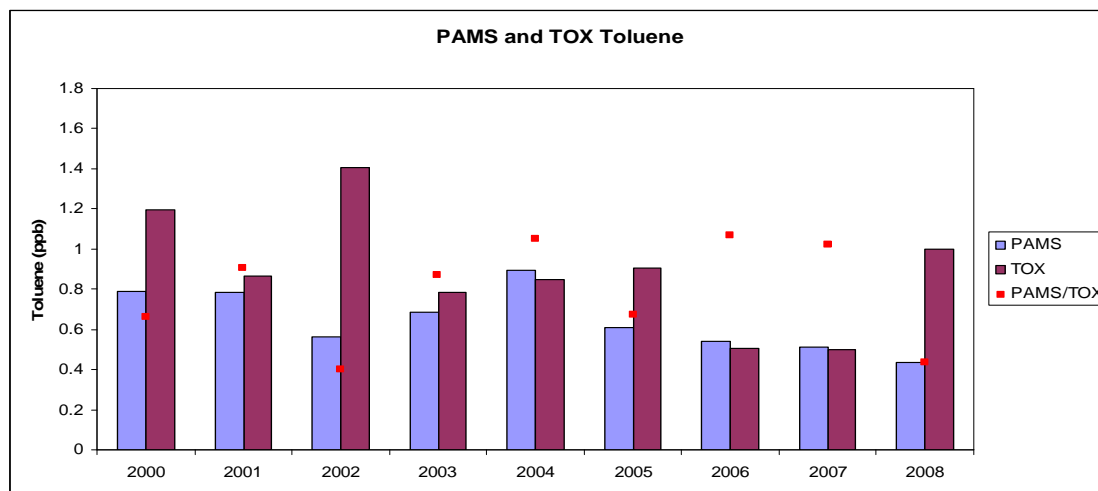
Note: Measurements were collected on the same days for collocated monitors. Only days with 75 percent data completeness (at least 18 hours) were included. Daily measurements are from the NJ Air Toxics site; hourly data from the PAMS network are averaged over the 24-hour period to match with the NJ Air Toxics data.

Figure A-10. Scatter plot of averaged PAMS and NJ Air Toxics measurements for toluene at Camden



Note: Measurements were collected on the same days for collocated monitors. Only days with 75 percent data completeness (at least 18 hours) were included. Daily measurements are from the NJ Air Toxics site; hourly data from the PAMS network are averaged over the 24-hour period to match with the NJ Air Toxics data. Two different methods of linear regression were used to develop trend lines, ordinary least squares (OLS) and least trimmed squares (LTS).

Figure A-11. Summer average PAMS and NJ Air Toxics measurements for toluene at Camden for 2000-2008

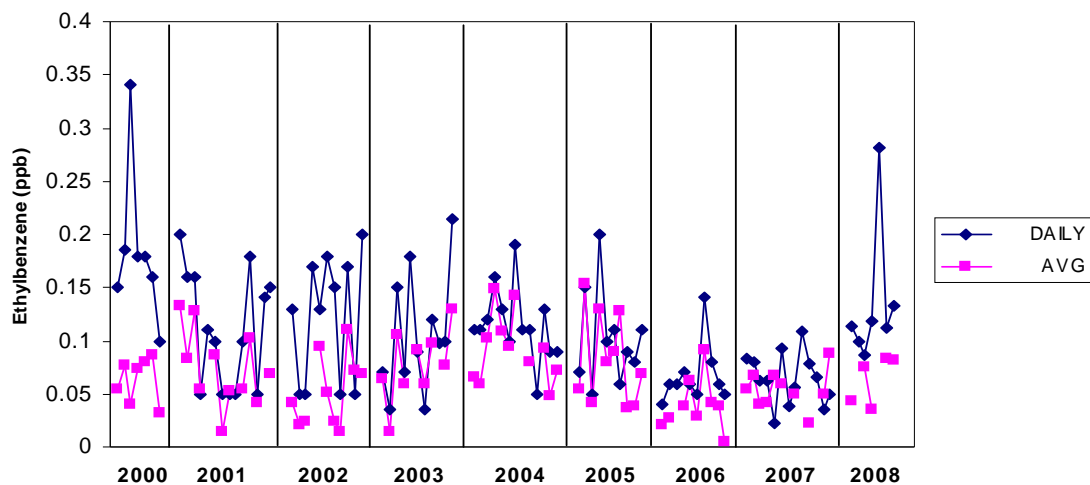


Note: Half of the minimum detection limit (MDL) was substituted for data points below the MDL, but no data completeness restrictions were imposed on the data used in the averages.

Ethylbenzene

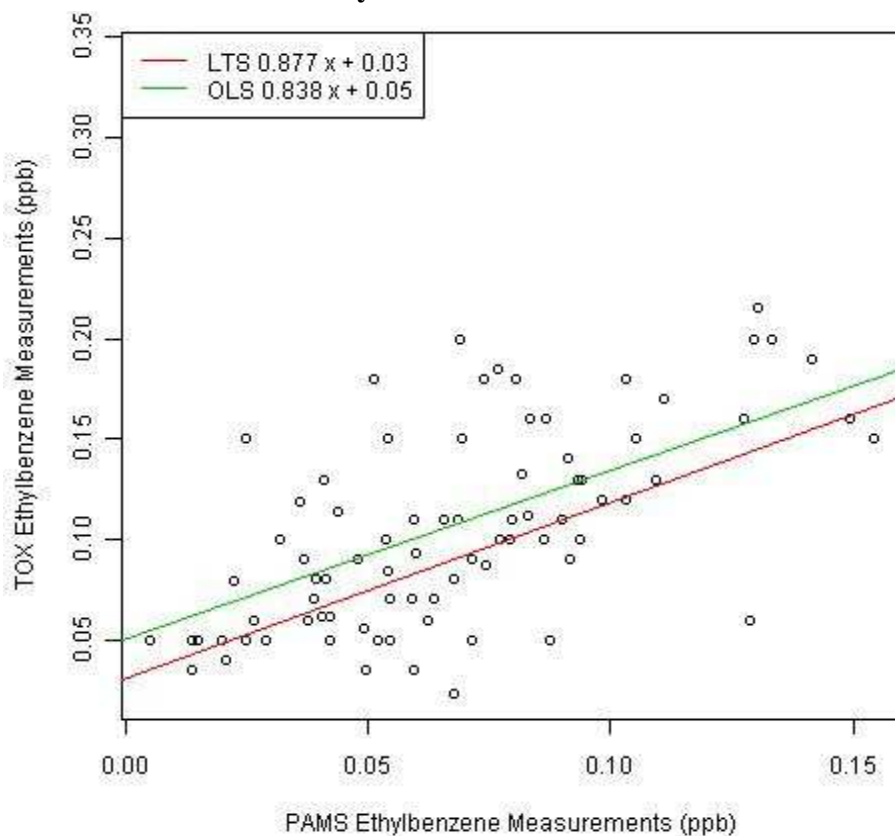
Figure A-12 and Figure A-13 contain time-series and scatter plots of ethylbenzene measurements for the NJ Air Toxics (blue line with diamonds) and PAMS (pink line with squares) samplers. For some years, the collocated and concurrent measurements of ethylbenzene do not match very well (e.g., 2000), while in others they do correspond in terms of absolute values and trends (e.g., 2004-2005). When all data are combined in a scatter plot (Figure A-13), there appears to be a linear relationship between the two types of data, although there is a significant amount of scatter. The slope is close to 1 (0.88 and 0.84) and the intercept is low for both OLS and LTS methods of linear regression, indicating that the daily ethylbenzene concentrations measured from the two different types of samplers are similar on average. Summer averages of ethylbenzene for both datasets also indicate that both datasets are comparable for most years (Figure A-14).

Figure A-12. Comparison of averaged PAMS and NJ Air Toxics measurements for ethylbenzene at Camden



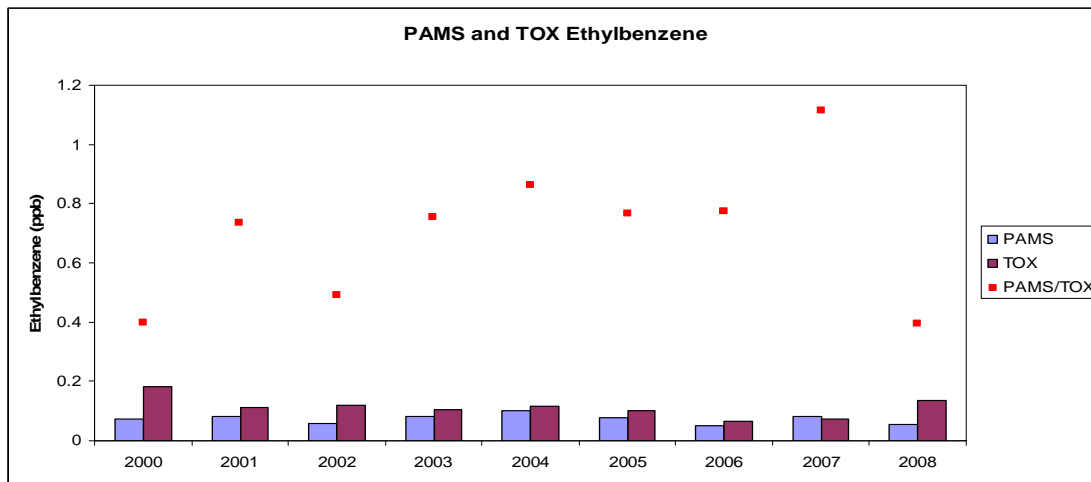
Note: Measurements were collected on the same days for collocated monitors. Only days with 75 percent data completeness (at least 18 hours) were included. Daily measurements are from the NJ Air Toxics site; hourly data from the PAMS network are averaged over the 24-hour period to match with the NJ Air Toxics data.

Figure A-13. Scatter plot of averaged PAMS and NJ Air Toxics measurements for ethylbenzene at Camden



Note: Measurements were collected on the same days for collocated monitors. Only days with 75 percent data completeness (at least 18 hours) were included. Daily measurements are from the NJ Air Toxics site; hourly data from the PAMS network are averaged over the 24-hour period to match with the NJ Air Toxics data. Two different methods of linear regression were used to develop trend lines, ordinary least squares (OLS) and least trimmed squares (LTS).

Figure A-14. Summer average PAMS and NJ Air Toxics measurements for ethylbenzene at Camden for 2000-2008



Note: Half of the minimum detection limit (MDL) was substituted for data points below the MDL, but no data completeness restrictions were imposed on the data used in the averages.

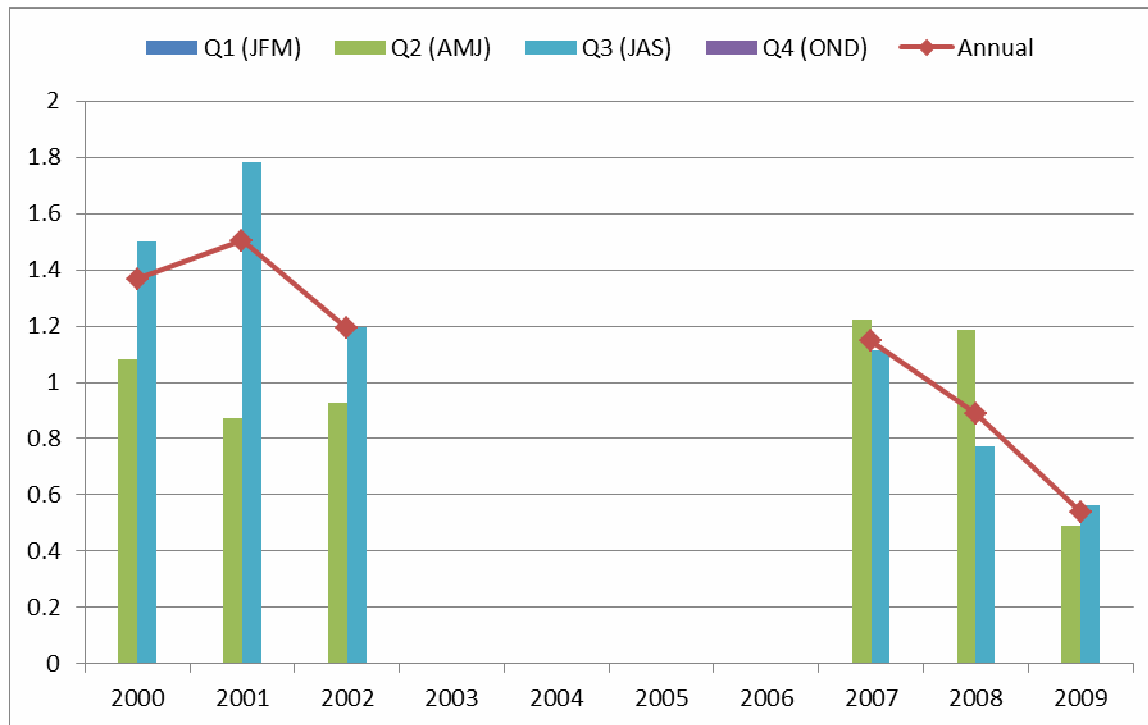
Appendix B: Regional air toxics measurements

Appendix B: Regional air toxics measurements

This appendix presents the results of NESCAUM's examination of regional air toxics measurements, as discussed in Section 3.4.4. Monitoring results for several species of interest are presented in this section in the same order as presented in Section 3.4. Results are presented for annual, quarterly, and diurnal averages, as available. For diurnal patterns, because sample sizes are generally small, single events (e.g., a plume with high air toxics concentrations) can have a large effect on the diurnal profile. These occurrences are noted when they occur in the diurnal profiles. Use of median concentrations rather than mean (simple average) concentrations would mitigate this effect.

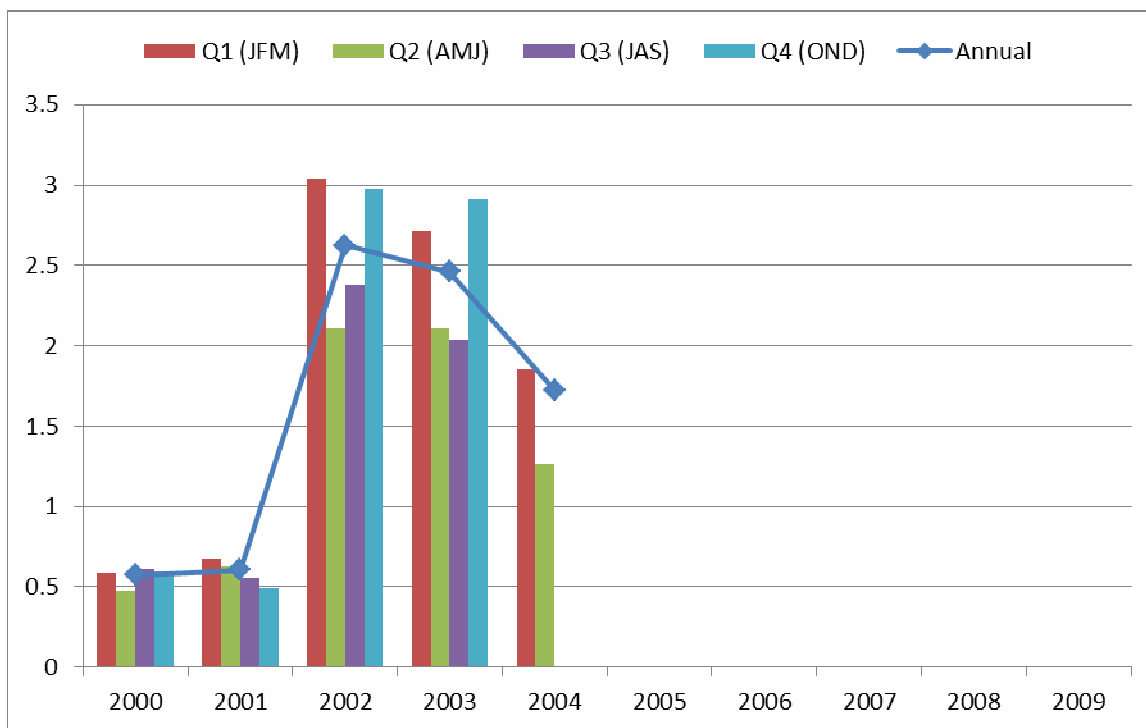
Benzene

Figure B-1. Quarterly and annual average benzene concentrations at Sherwood Island, Connecticut



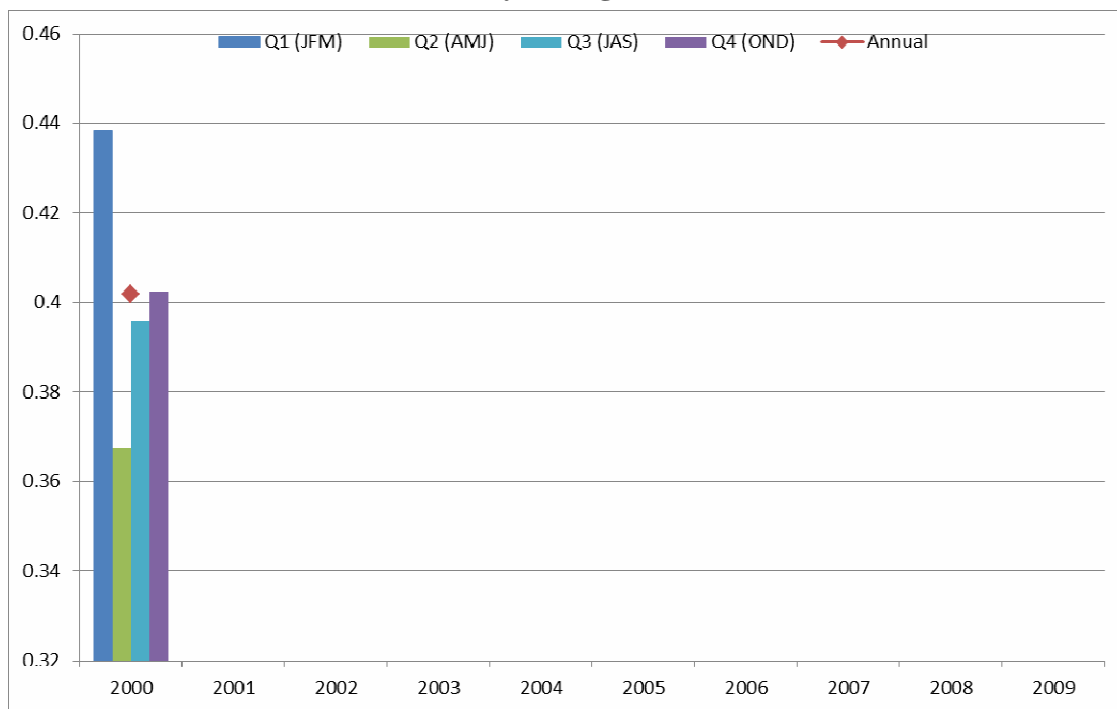
Note: Data were not available for Q1, Q4, or years 2003-2006 at this location. Sherwood Island, Connecticut has only PAMS data; no 24-hour year-round data are available at this site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-2. Quarterly and annual average benzene concentrations at Botanical Gardens, NY



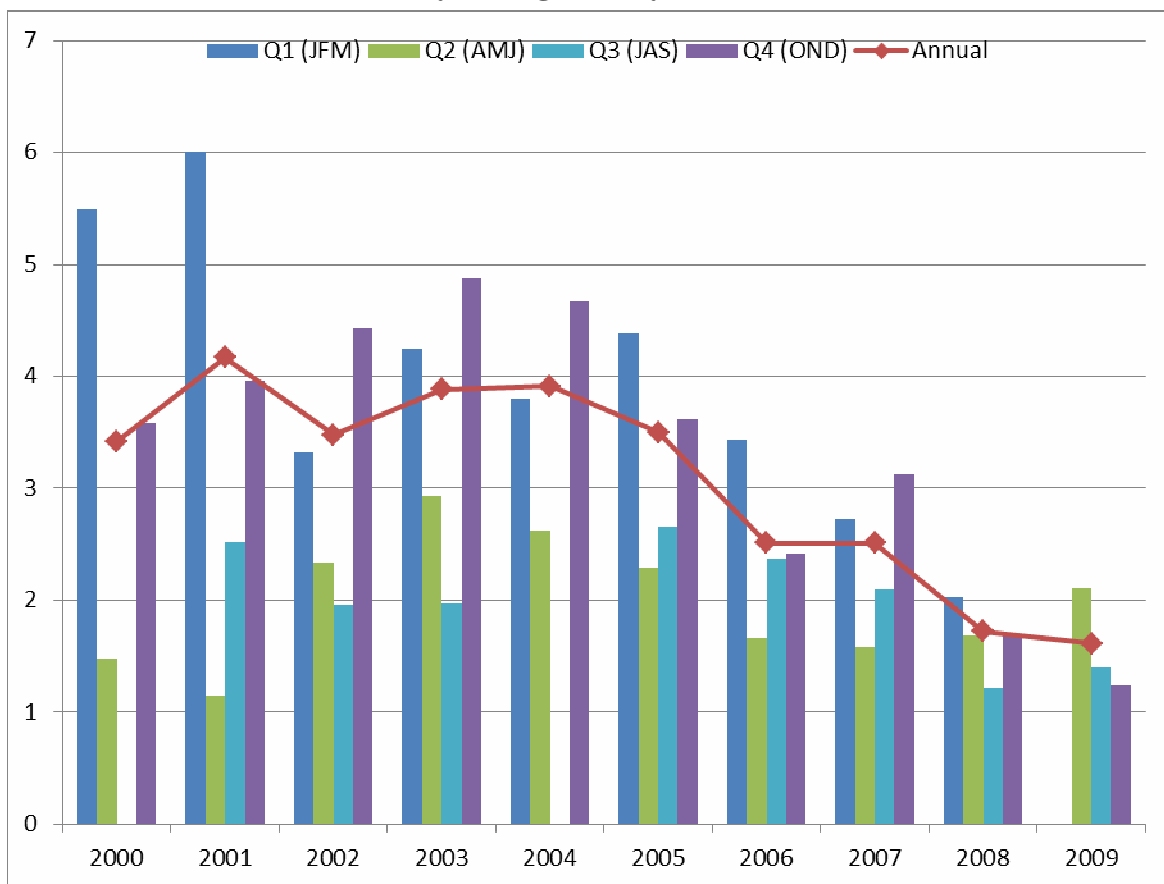
Note: Data were not available for Q3 2004 to present at this location. Data presented are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-3. Quarterly and annual average benzene concentrations at Queens Community College, New York



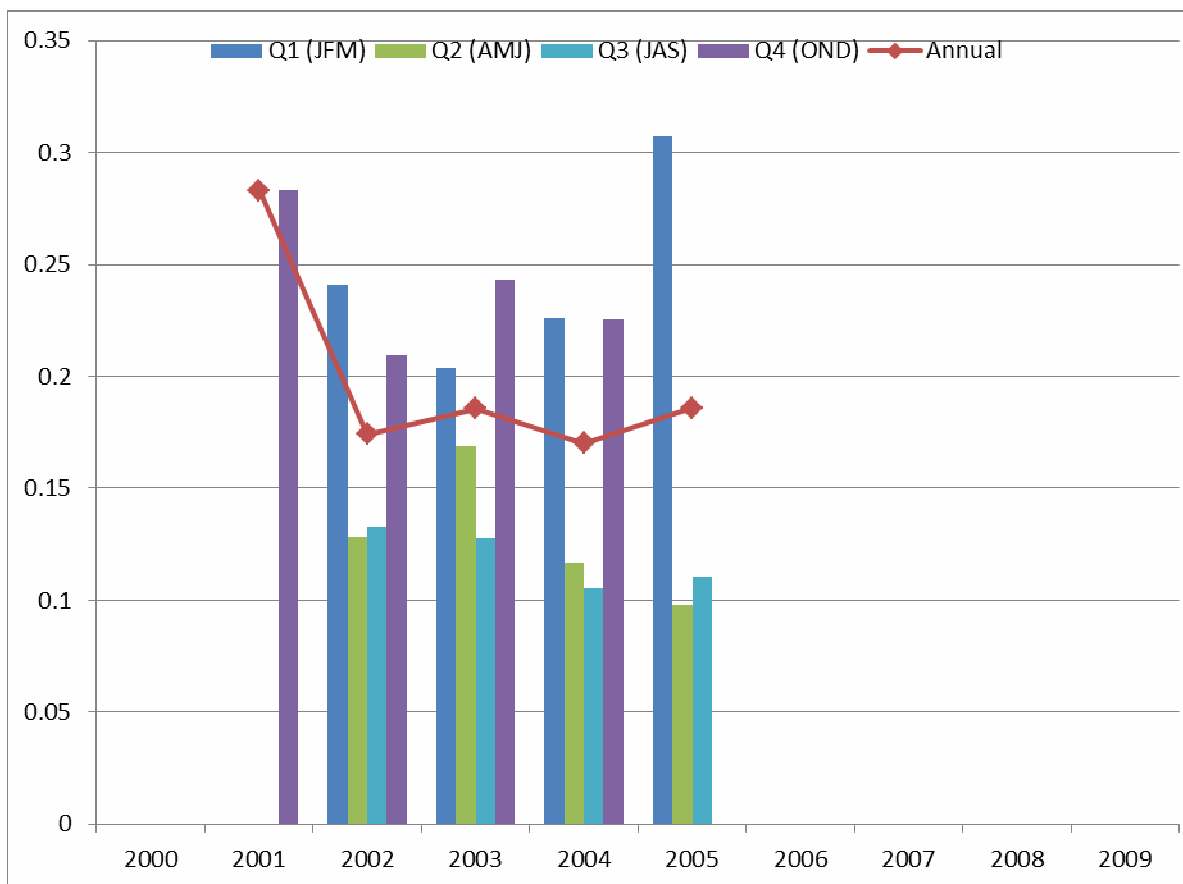
Note: Data were not available for 2001 to present at this location. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-4. Quarterly and annual average benzene concentrations at East Lycoming, Pennsylvania



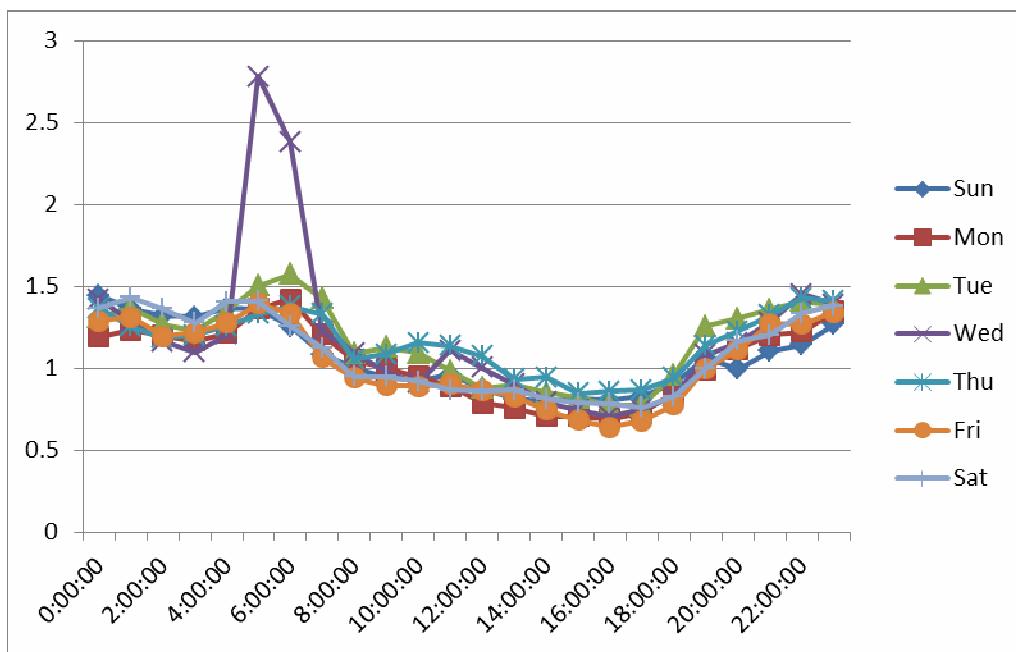
Note: Data were not available for Q3 2000 or Q3 2004. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-5. Quarterly and annual average benzene concentrations at Lums Pond, Delaware



Note: The site became active in Q4 2001 and stopped taking air toxics measurements in Q4 2005. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-6. Comparison of average hourly benzene concentrations at the Sherwood Island, Connecticut PAMS



Note: The peak for hours 5-6 on Wednesday is due to several events where monitored concentrations were much higher than typical for those times.

Figure B-7. Comparison of average hourly benzene concentrations at the Botanical Gardens, New York PAMS

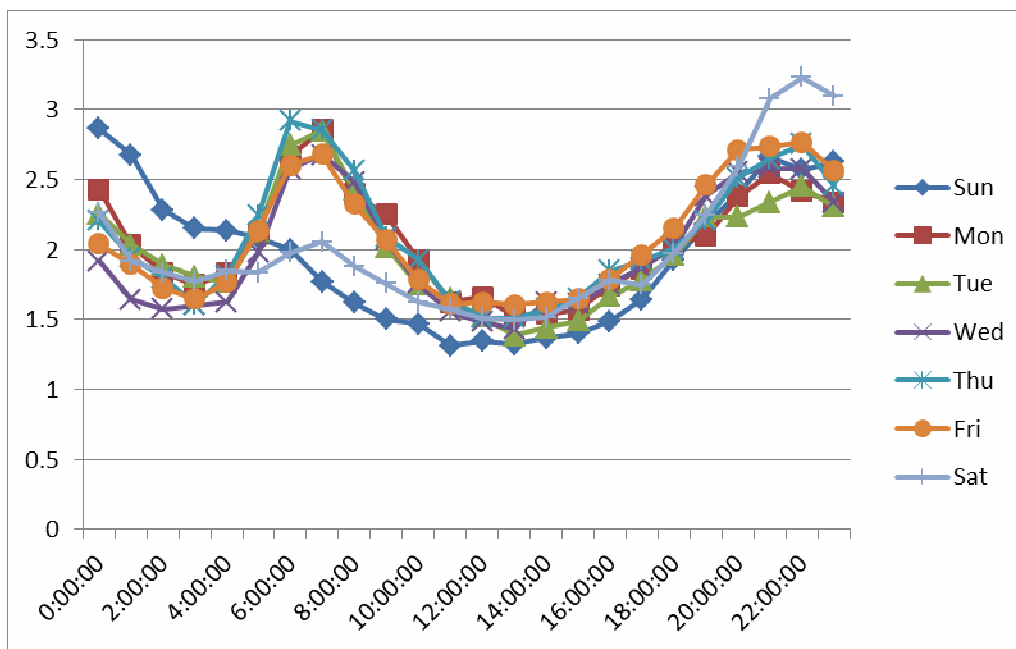


Figure B-8. Comparison of average hourly benzene concentrations at the Queens Community College, New York PAMS

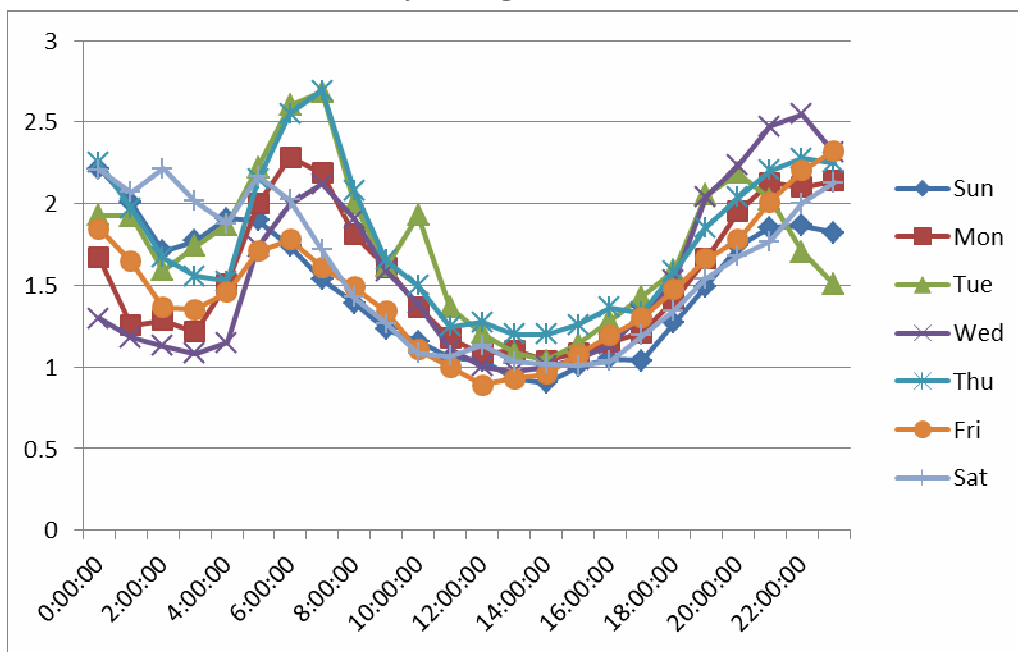


Figure B-9. Comparison of average hourly benzene concentrations at the East Lycoming, Pennsylvania PAMS

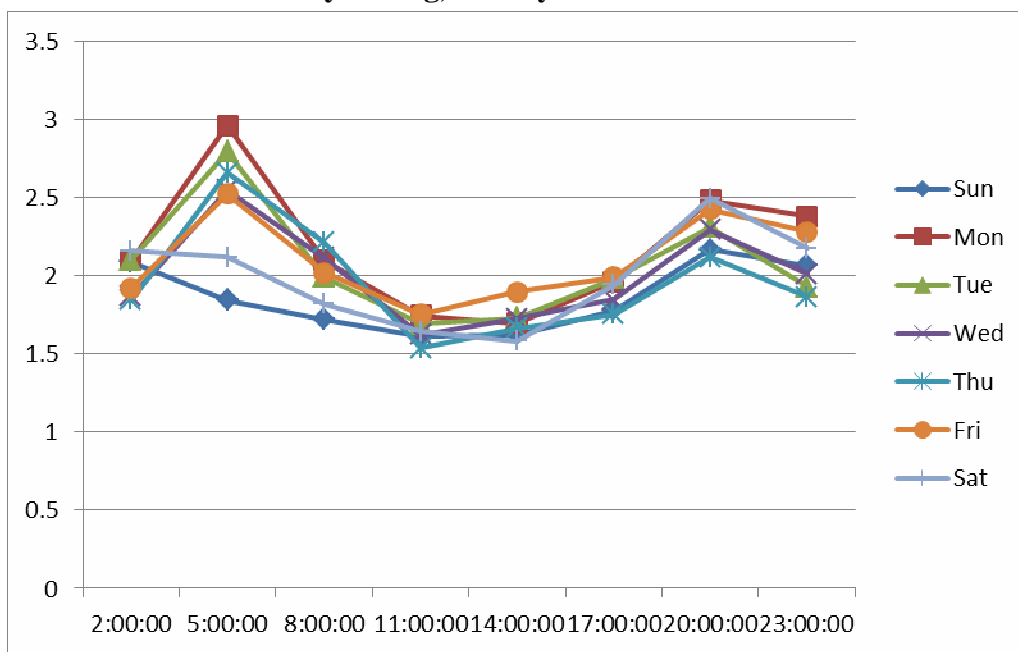
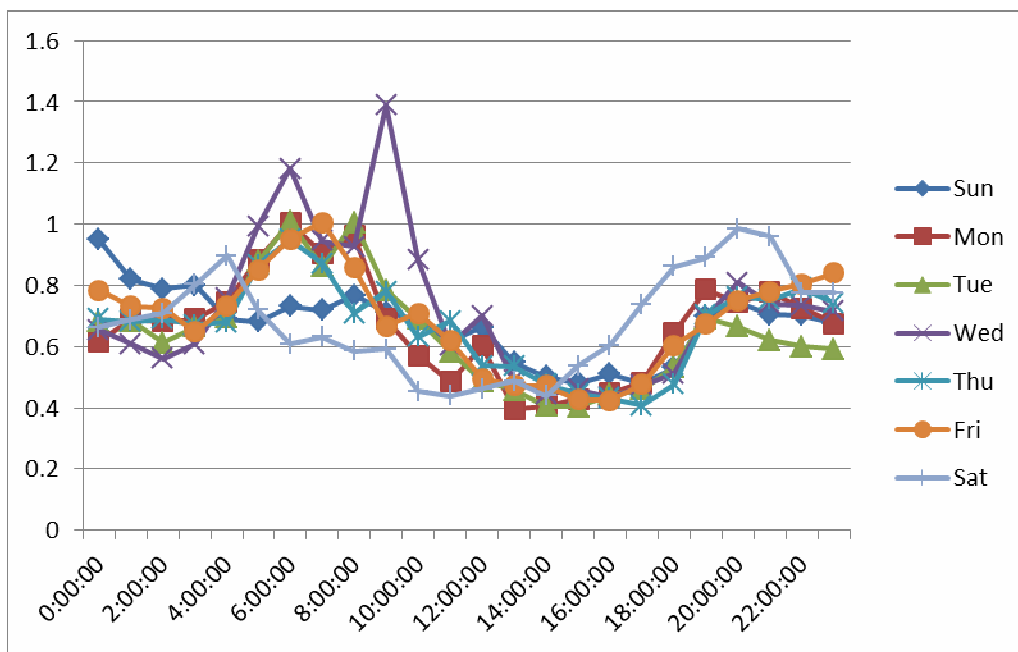
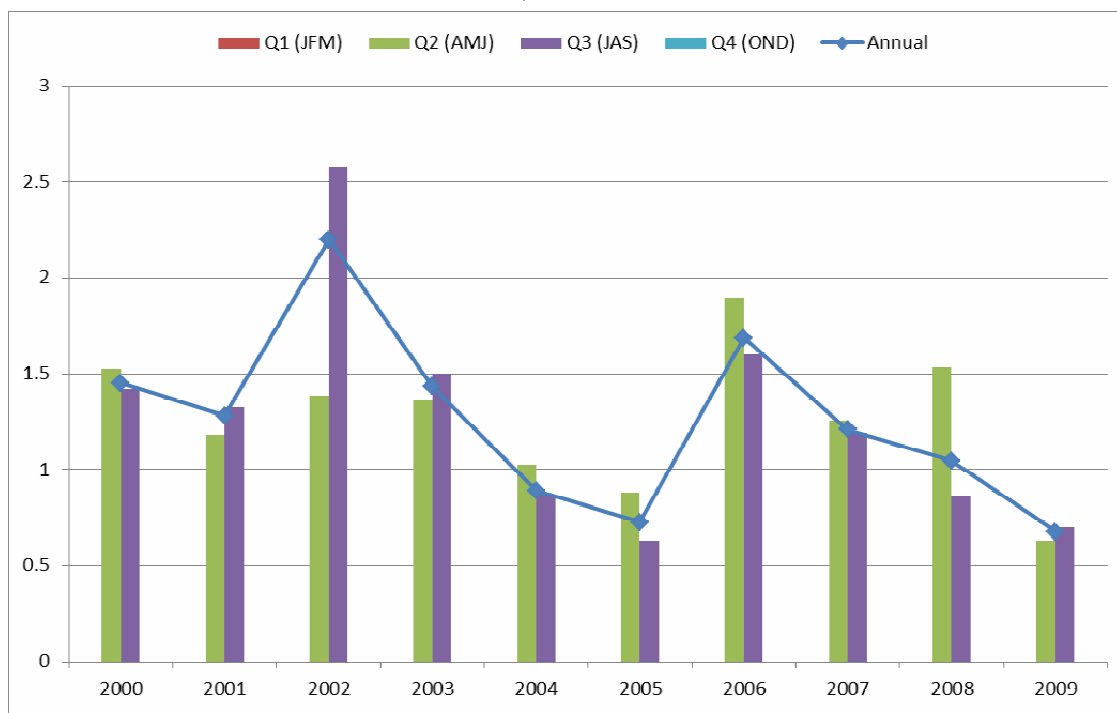


Figure B-10. Comparison of average hourly benzene concentrations at the Lums Pond, Delaware PAMS

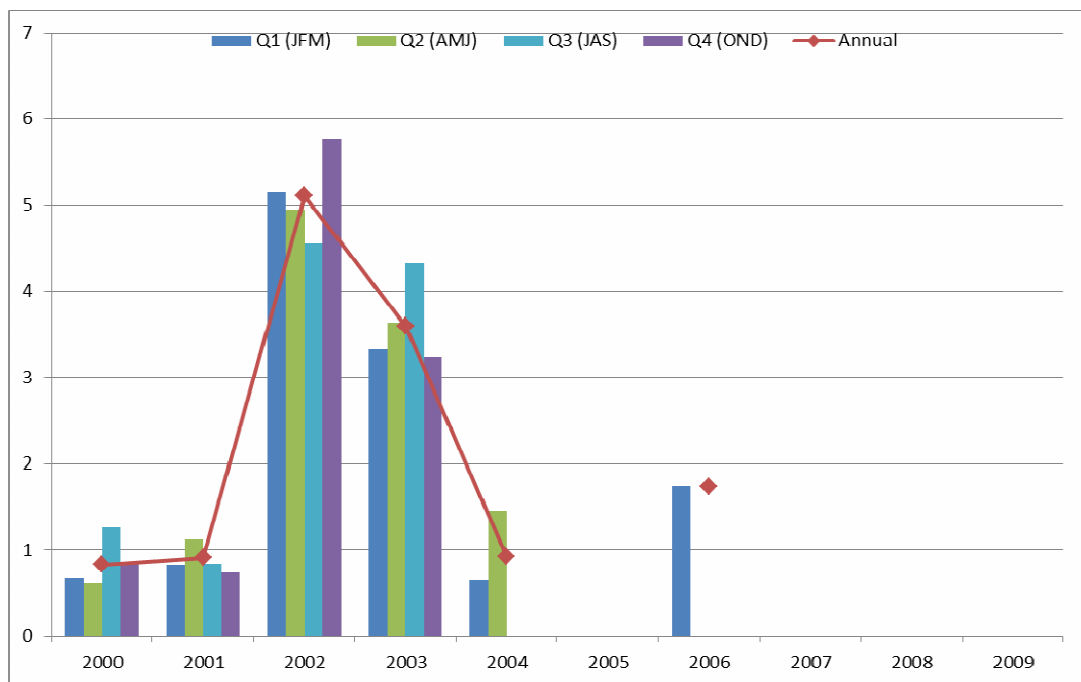


Note: The peak for hours 6 and 9 on Wednesday are primarily due to single events where monitored concentrations were much higher than typical for those time.

m,p-Xylene**Figure B-11. Quarterly and annual average *m,p*-xylene concentrations at Sherwood Island, Connecticut**

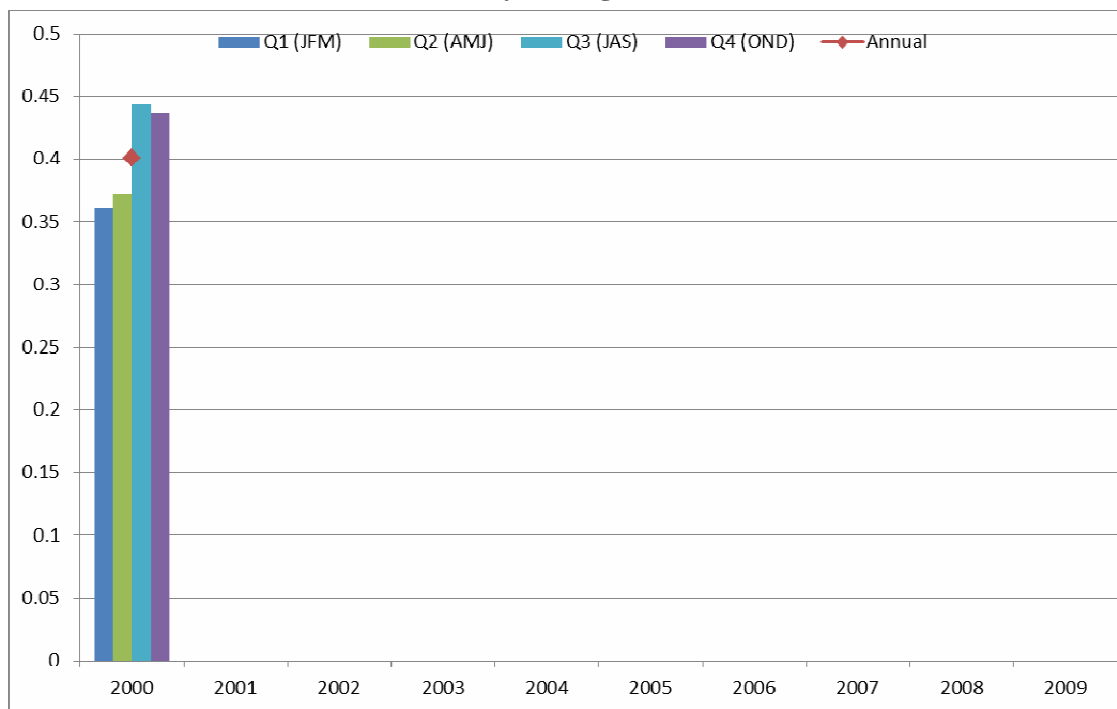
Note: Data were not available for Q1, Q4 at this location. Sherwood Island, Connecticut has only PAMS data; no 24-hour year-round data are available at this site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-12. Quarterly and annual average *m,p*-xylene concentrations at Botanical Gardens, New York



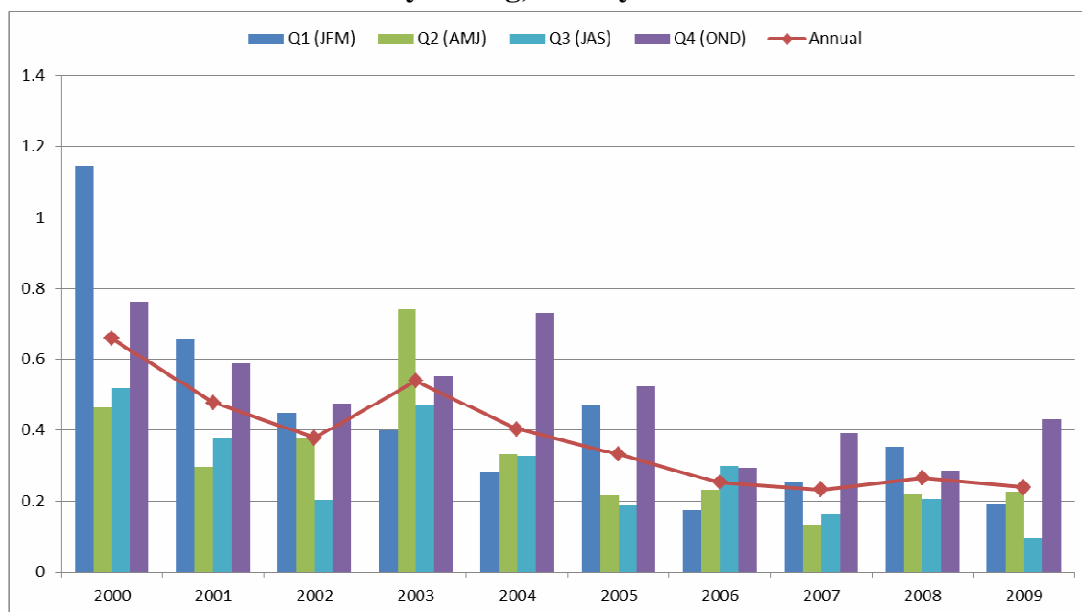
Note: Data were not available for Q3 2004 to present at this location, except during Q1 2006. Data presented are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-13. Quarterly and annual average *m,p*-xylene concentrations at Queens Community College, New York



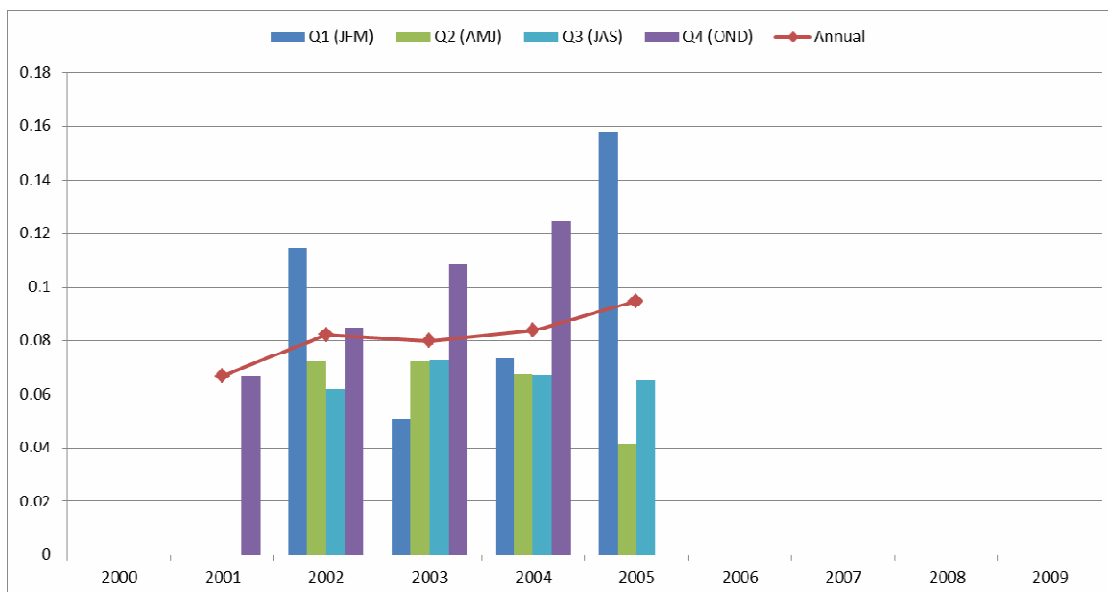
Note: Data were not available for 2001 to present at this location. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-14. Quarterly and annual average *m,p*-xylene concentrations at East Lycoming, Pennsylvania



Note: Data were not available for Q3 2000. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-15. Quarterly and annual average *m,p*-xylene concentrations at Lums Pond, Delaware



Note: The site became active in Q4 2001 and stopped taking air toxics measurements in Q4 2005. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-16. Comparison of average hourly *m,p*-xylene concentrations at the Sherwood Island, Connecticut PAMS

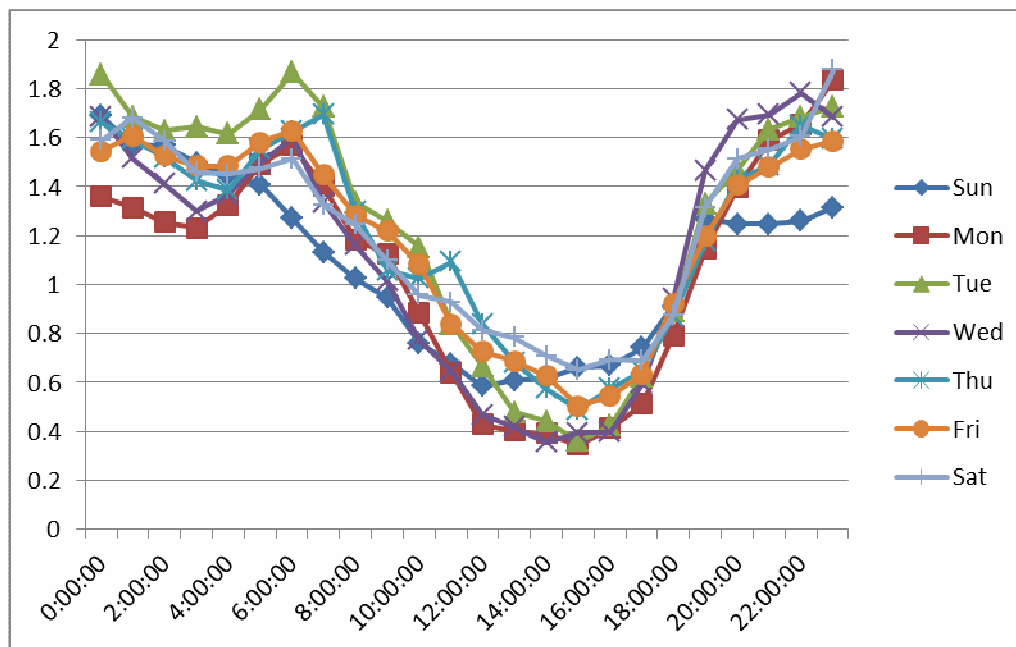


Figure B-17. Comparison of average hourly *m,p*-xylene concentrations at the Botanical Gardens, New York PAMS

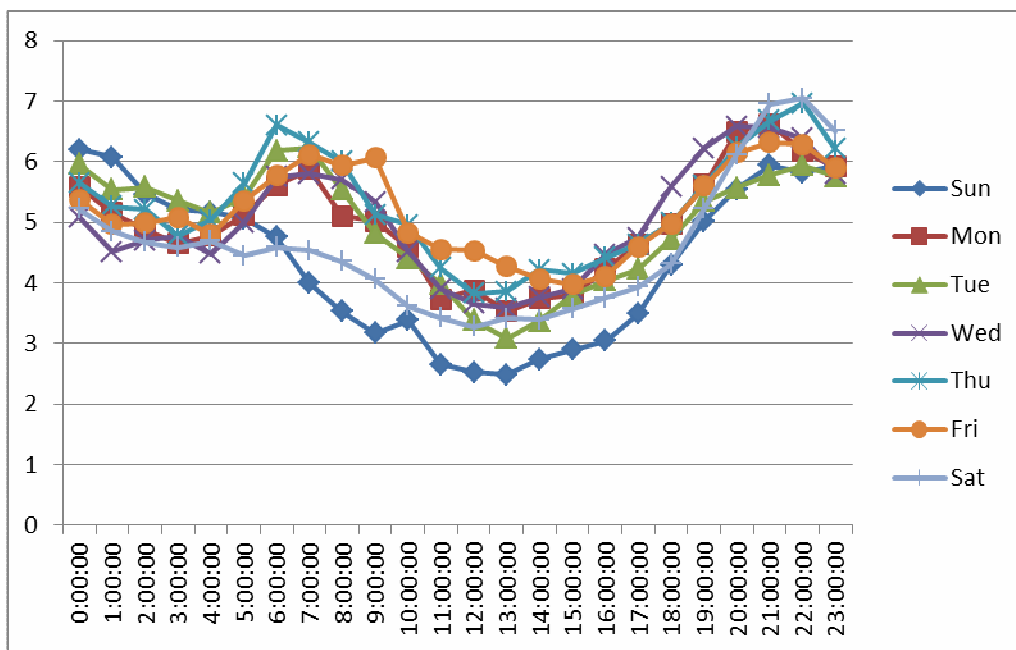
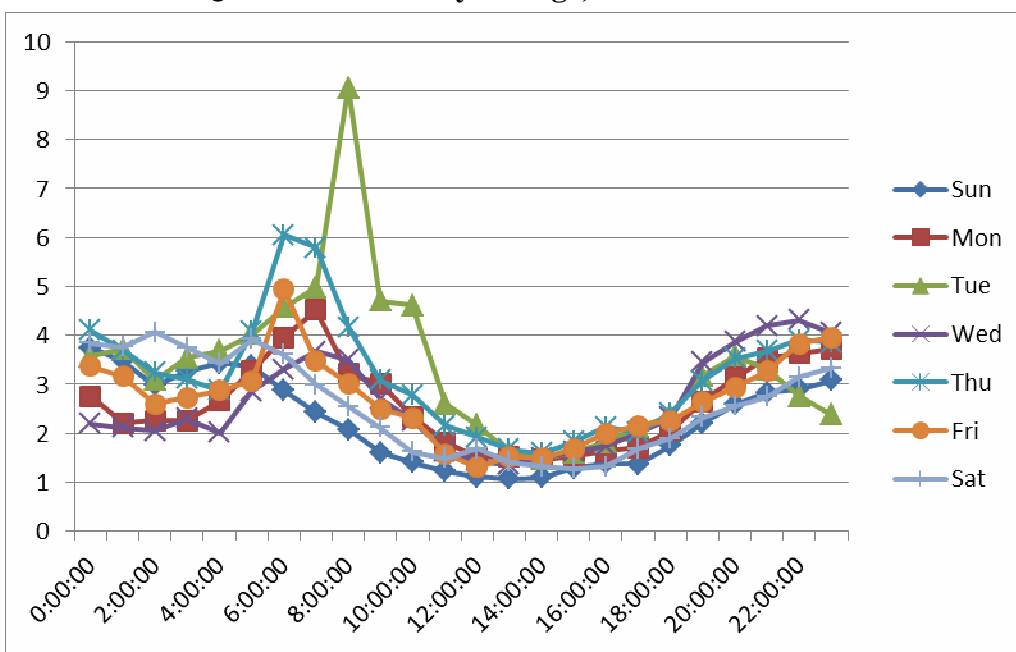


Figure B-18. Comparison of average hourly *m,p*-xylene concentrations at the Queens Community College, New York PAMS



Note: The peak for hours 8-10 on Tuesday is due to a single event where monitored concentrations were much higher than typical for those times.

Figure B-19. Comparison of average hourly *m,p*-xylene concentrations at the East Lycoming, Pennsylvania PAMS

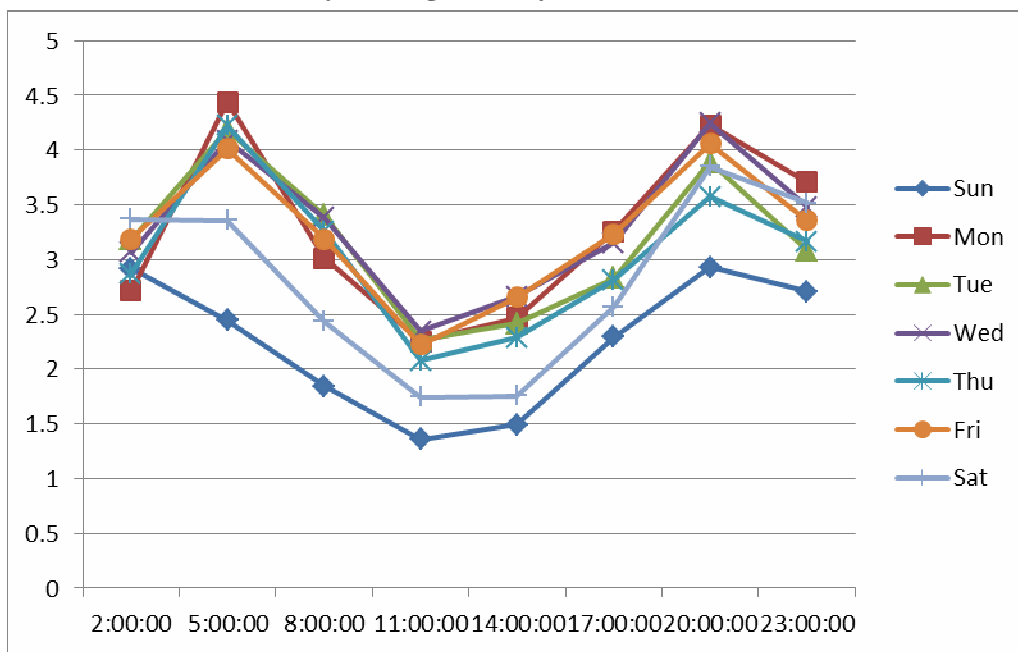
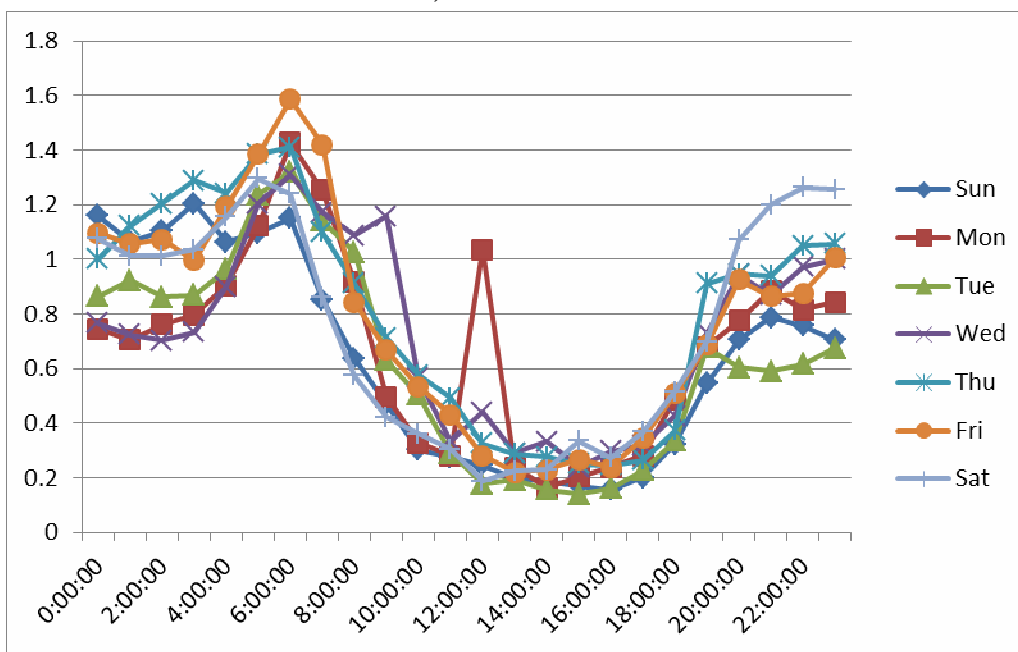


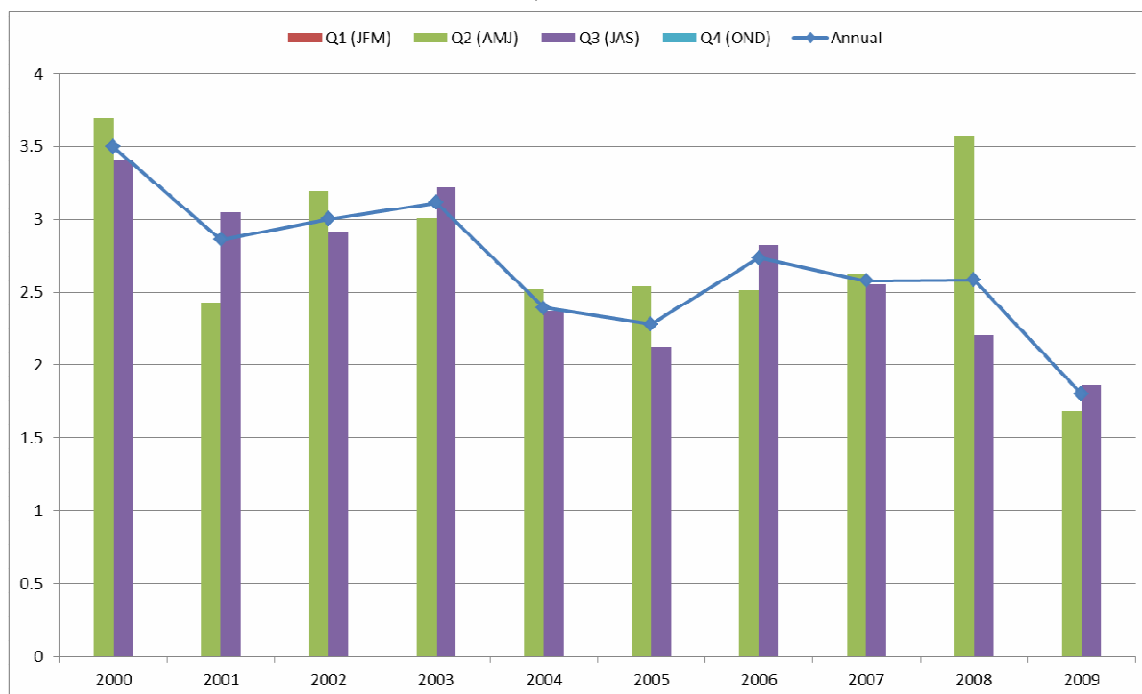
Figure B-20. Comparison of average hourly *m,p*-xylene concentrations at the Lums Pond, Delaware PAMS



Note: The peak for hour 12 on Monday is due to a single event where the monitored concentration was much higher than any other time (more than three times the next highest observed concentration).

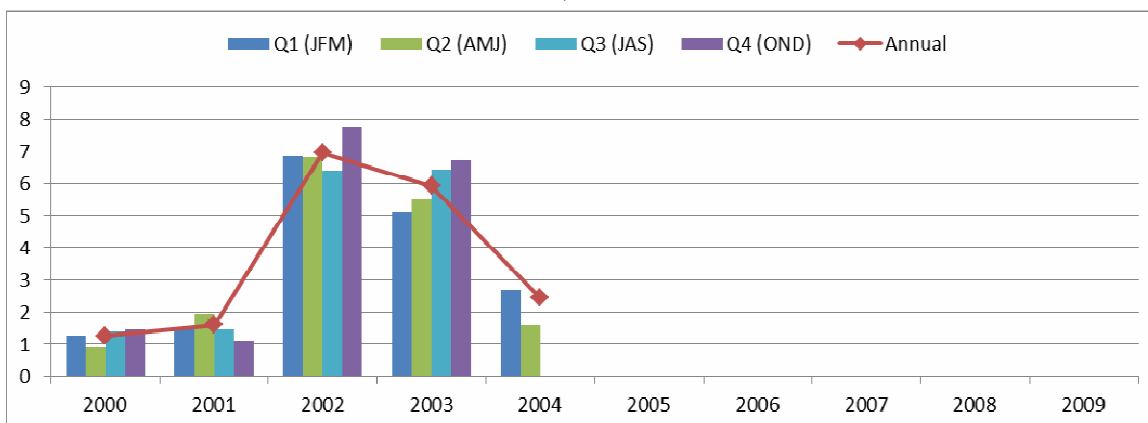
Toluene

Figure B-21. Quarterly and annual average toluene concentrations at Sherwood Island, Connecticut



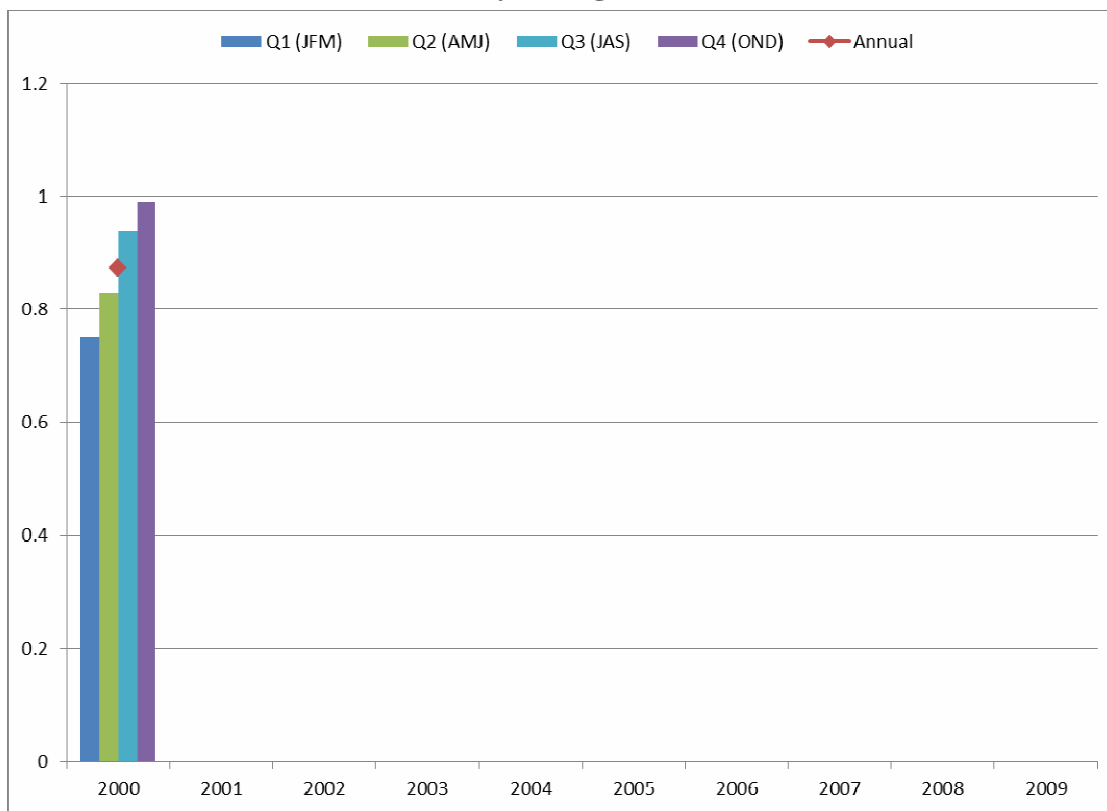
Note: Data were not available for Q1, Q4 at this location. Sherwood Island, Connecticut has only PAMS data; no 24-hour year-round data are available at this site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-22. Quarterly and annual average toluene concentrations at Botanical Gardens, New York



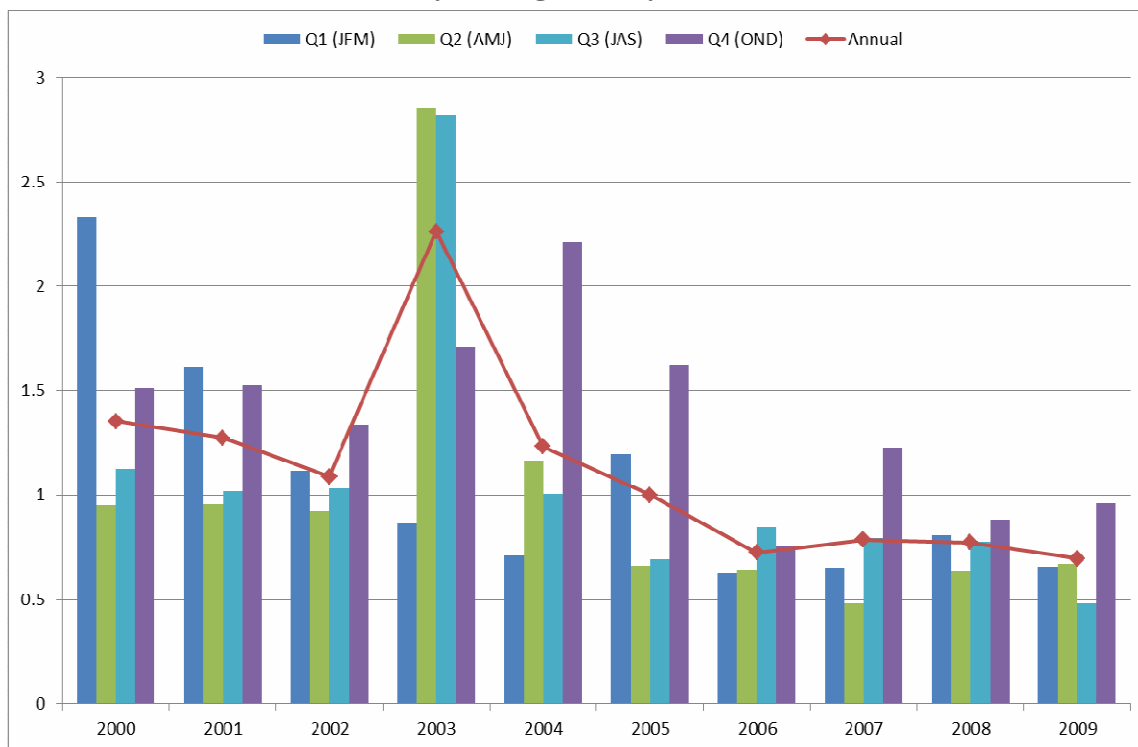
Note: Data were not available for Q3 2004 to present at this location. Data presented are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-23. Quarterly and annual average toluene concentrations at Queens Community College, New York



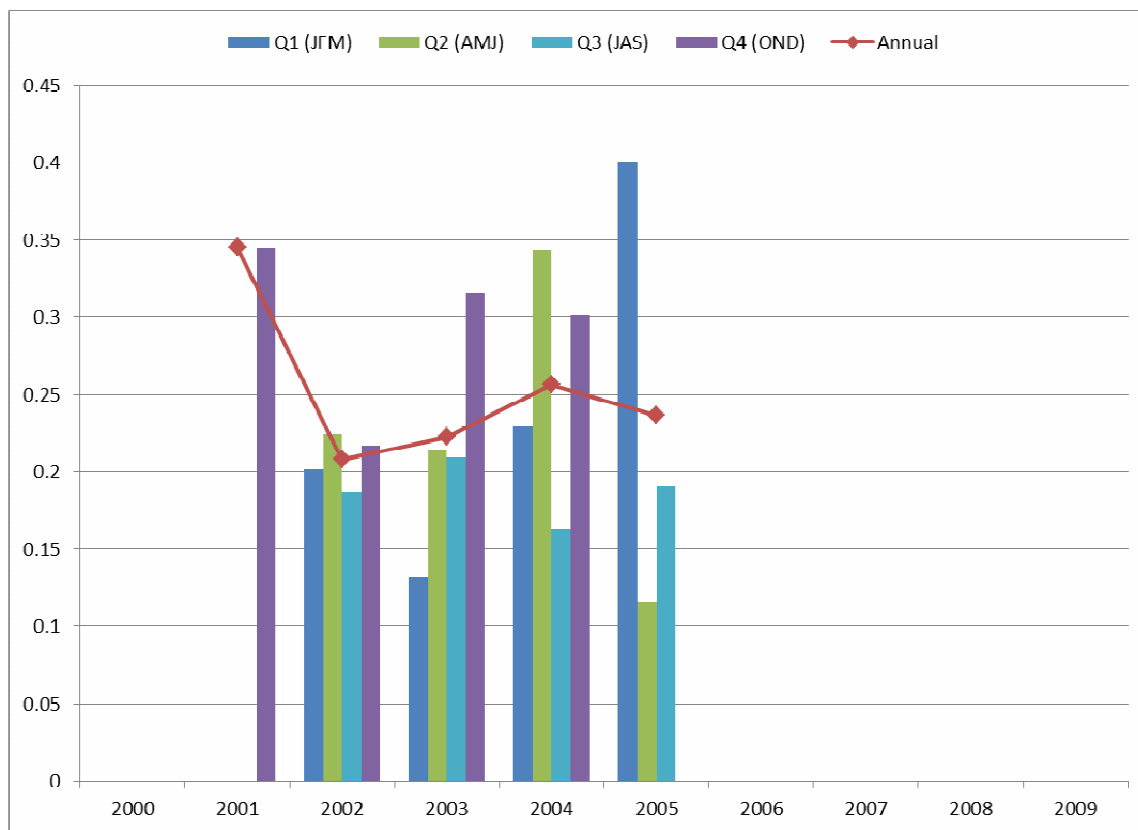
Note: Data were not available for 2001 to present at this location. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-24. Quarterly and annual average toluene concentrations at East Lycoming, Pennsylvania



Note: Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-25. Quarterly and annual average toluene concentrations at Lums Pond, Delaware



Note: The site became active in Q4 2001 and stopped taking air toxics measurements in Q4 2005. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-26. Comparison of average hourly toluene concentrations at the Sherwood Island, Connecticut PAMS

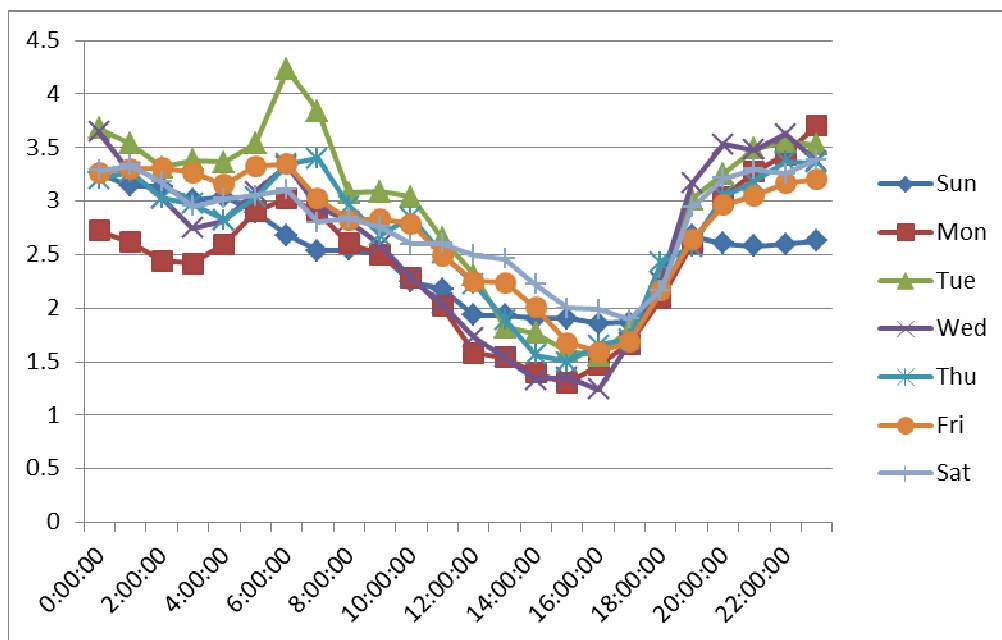
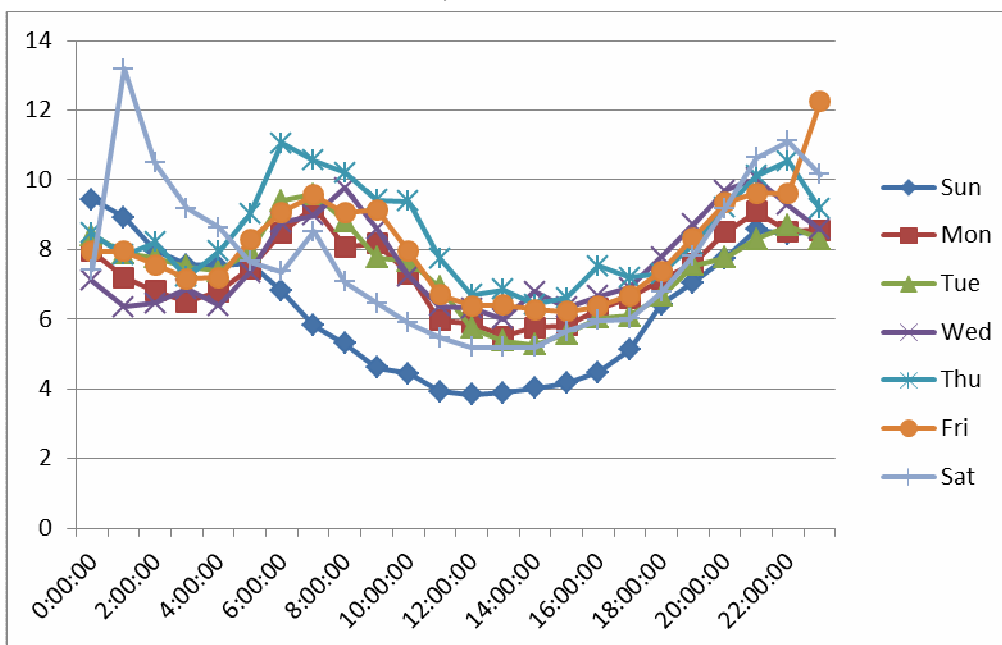
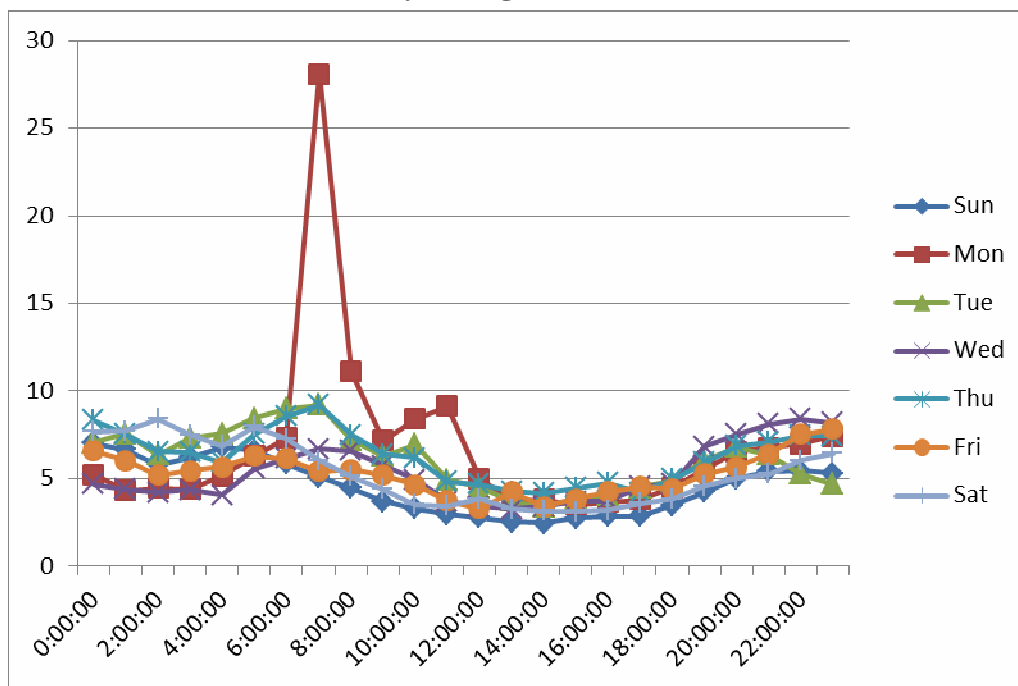


Figure B-27. Comparison of average hourly toluene concentrations at the Botanical Gardens, New York PAMS



Note: The peaks for hour 23 on Friday and hours 1-5 on Saturday are due to a single event where monitored concentrations were much higher than typical for those times.

Figure B-28. Comparison of average hourly toluene concentrations at the Queens Community College, New York PAMS



Note: The peaks for hours 7-11 on Monday are due primarily to a single event where the monitored concentrations were much higher than typical for those times.

Figure B-29. Comparison of average hourly toluene concentrations at the East Lycoming, Pennsylvania PAMS

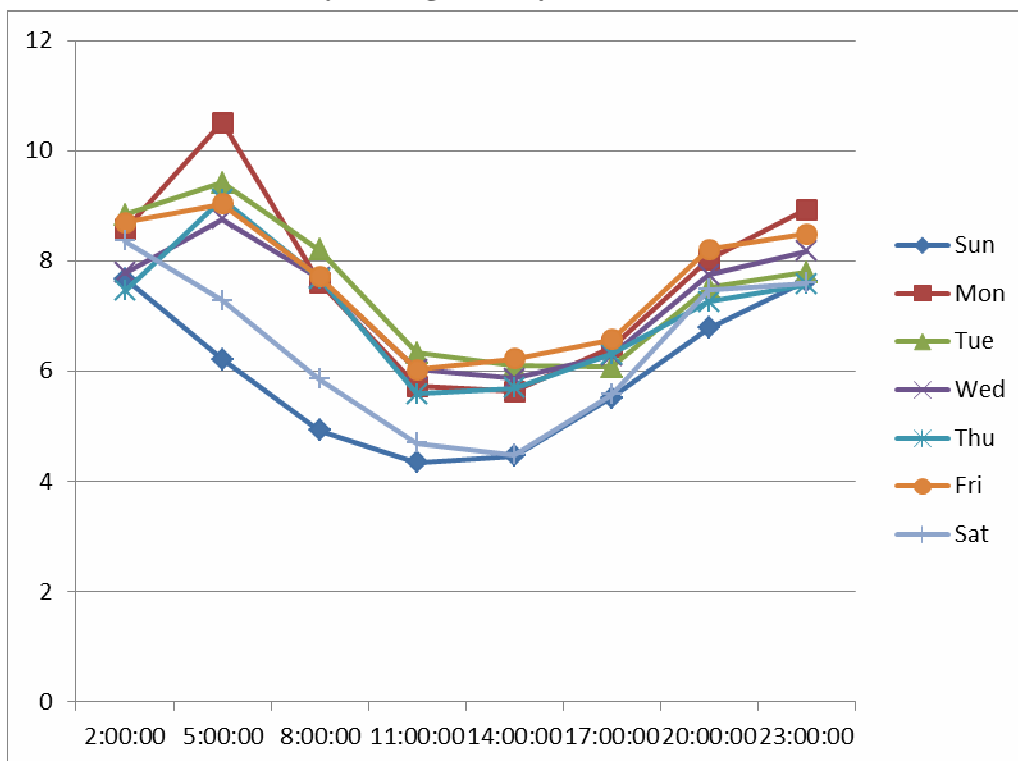
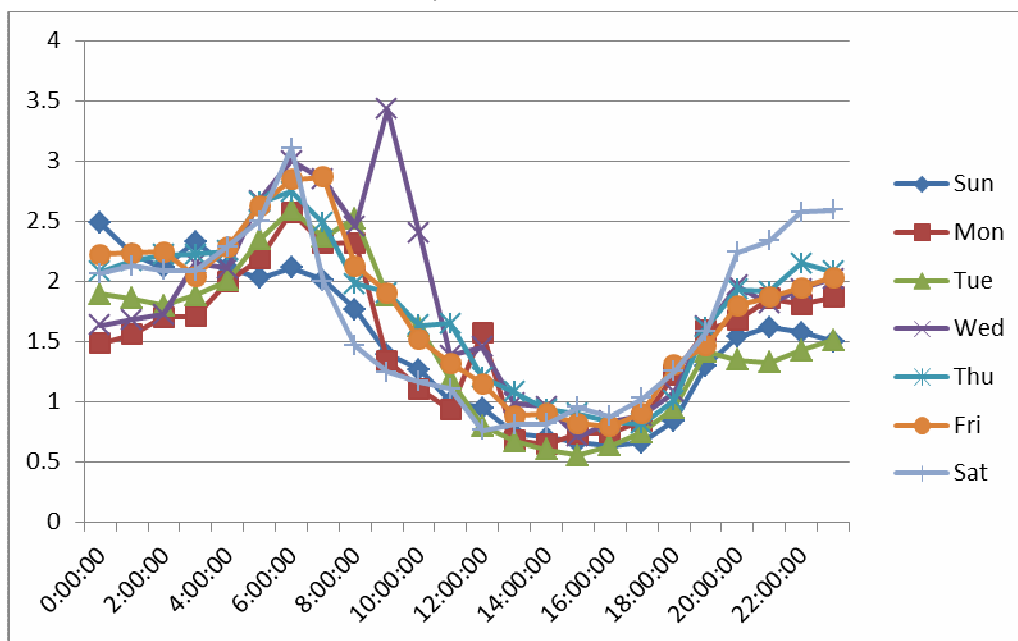
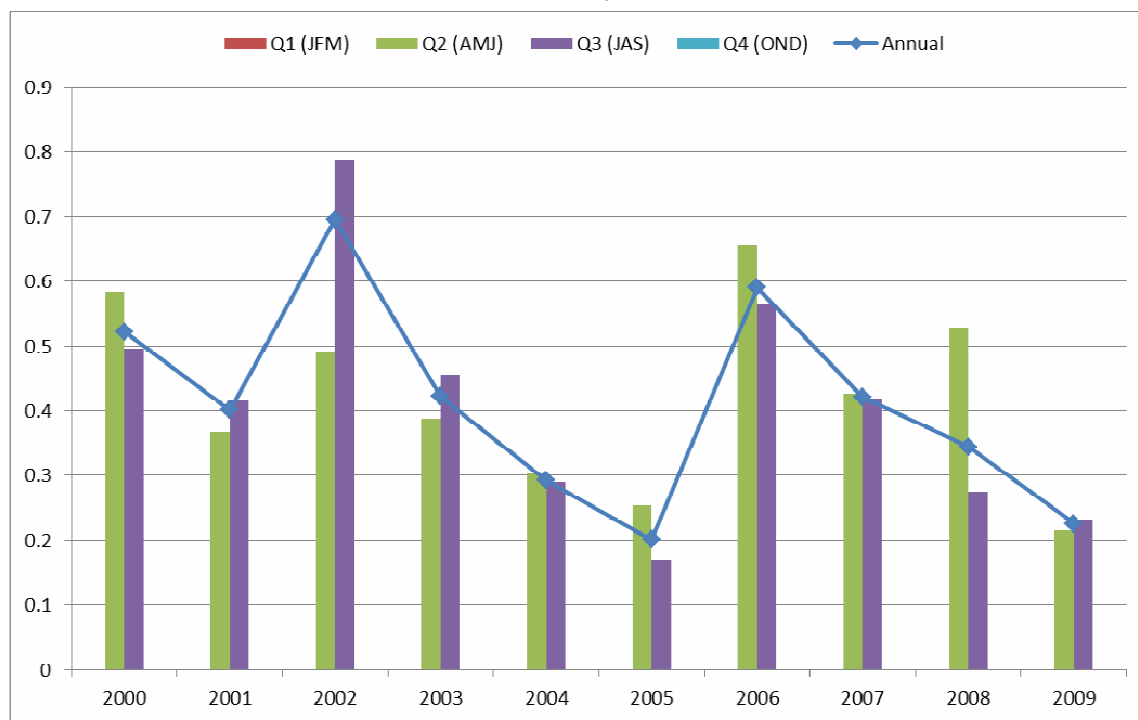


Figure B-30. Comparison of average hourly toluene concentrations at the Lums Pond, Delaware PAMS

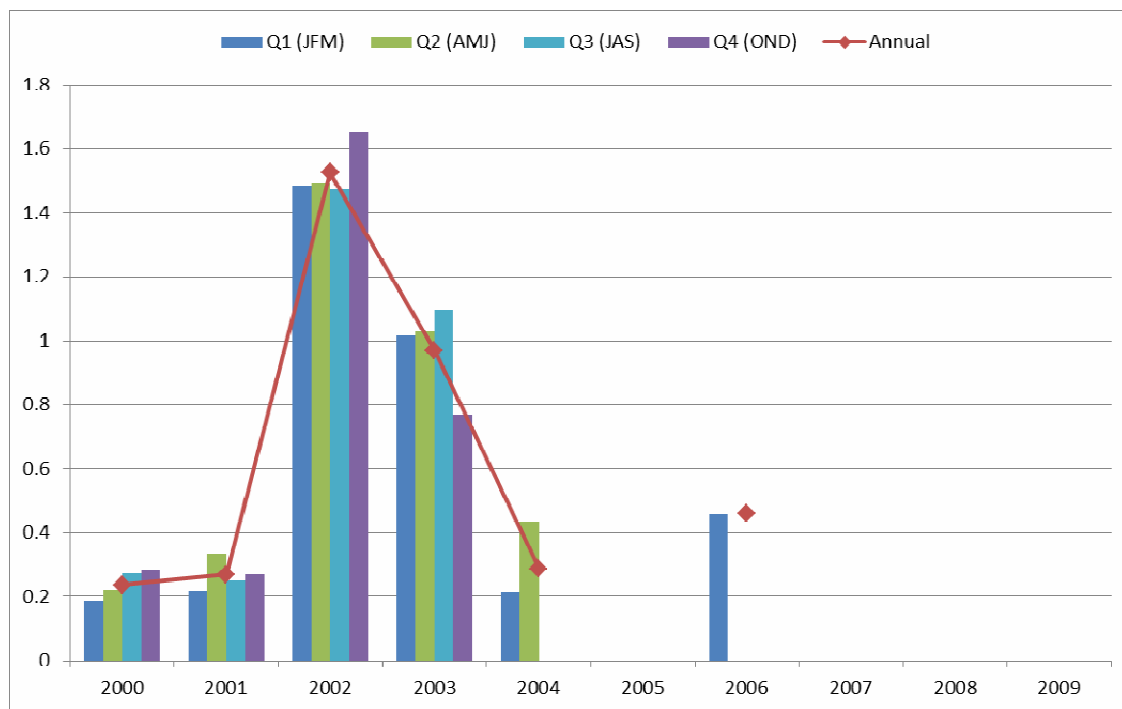


Note: The peaks for hours 9-10 on Wednesday are due to several events where the monitored concentrations were higher than typical for those times.

*Ethylbenzene***Figure B-31. Quarterly and annual average ethylbenzene concentrations at Sherwood Island, Connecticut**

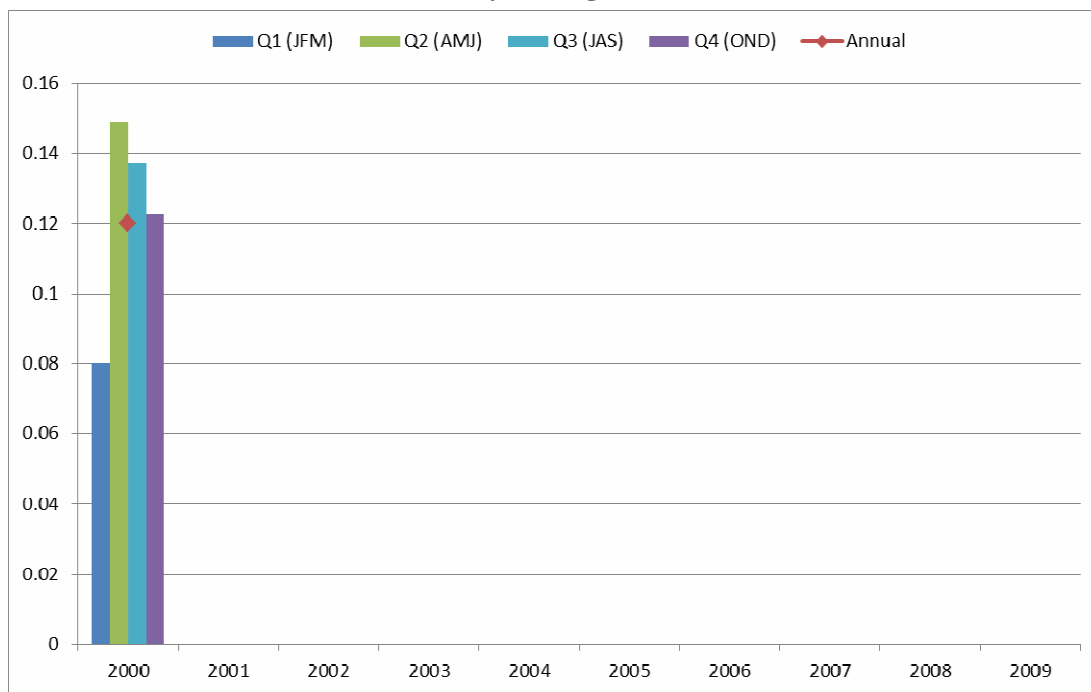
Note: Data were not available for Q1, Q4 at this location. Sherwood Island, Connecticut has only PAMS data; no 24-hour year-round data are available at this site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-32. Quarterly and annual average ethylbenzene concentrations at Botanical Gardens, New York



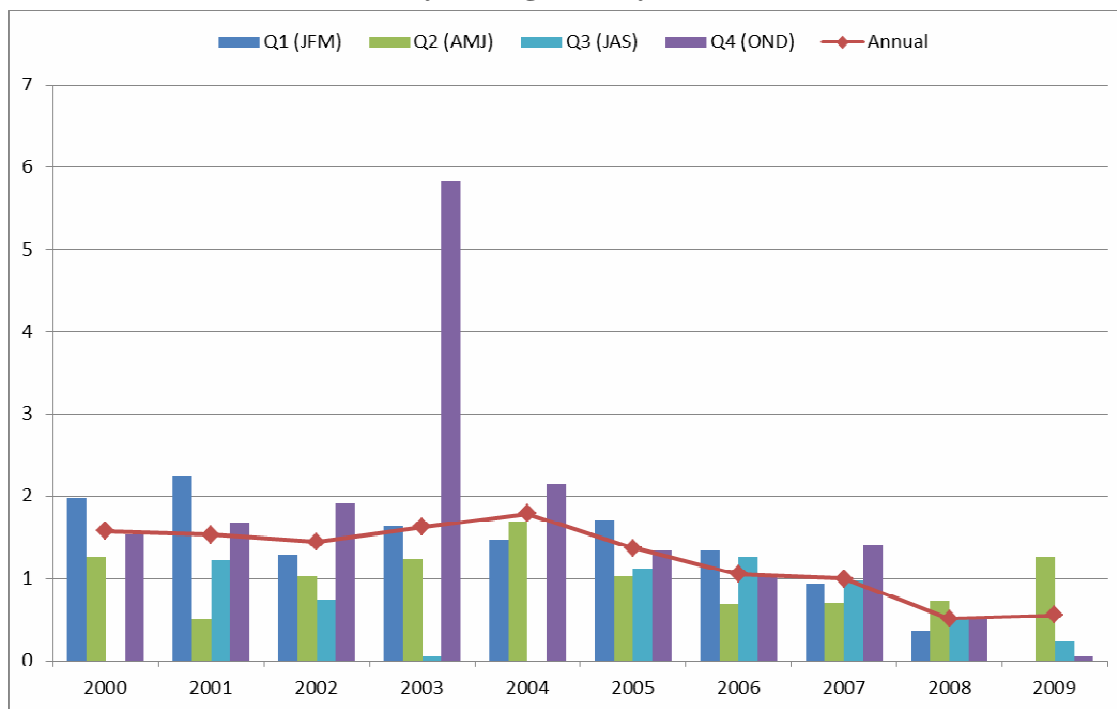
Note: Data were not available for Q3 2004 to present except for Q1 2006 at this location. Data presented are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-33. Quarterly and annual average ethylbenzene concentrations at Queens Community College, New York



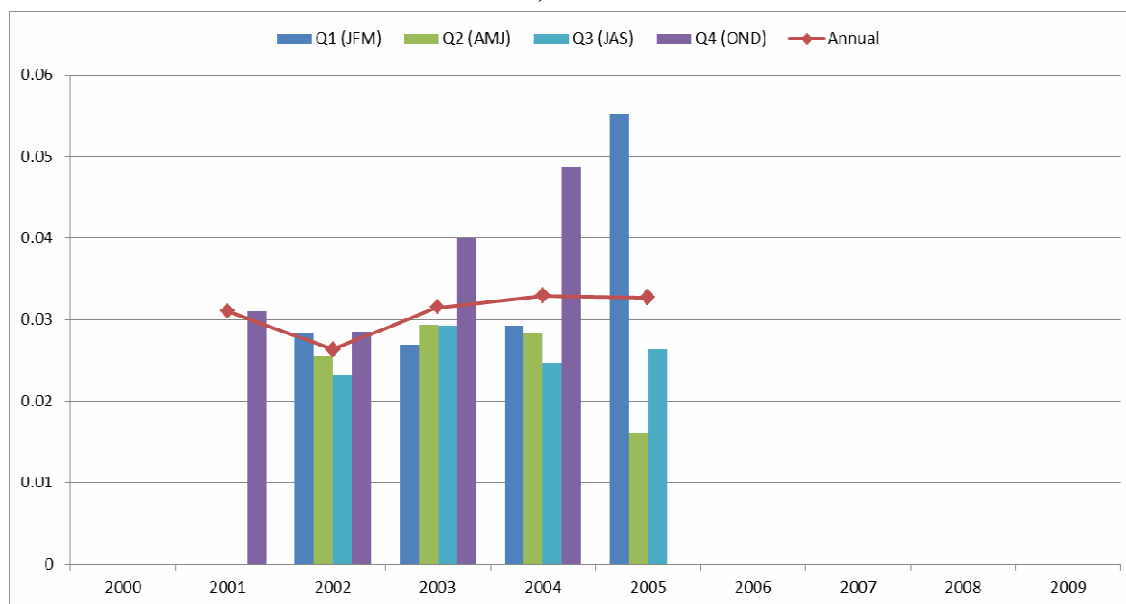
Note: Data were not available for 2001 to present at this location. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-34. Quarterly and annual average ethylbenzene concentrations at East Lycoming, Pennsylvania



Note: Data were not available for Q3 2000 or Q3 2004. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-35. Quarterly and annual average ethylbenzene concentrations at Lums Pond, Delaware



Note: The site became active in Q4 2001 and stopped taking air toxics measurements in Q4 2005. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-36. Comparison of average hourly ethylbenzene concentrations at the Sherwood Island, Connecticut PAMS

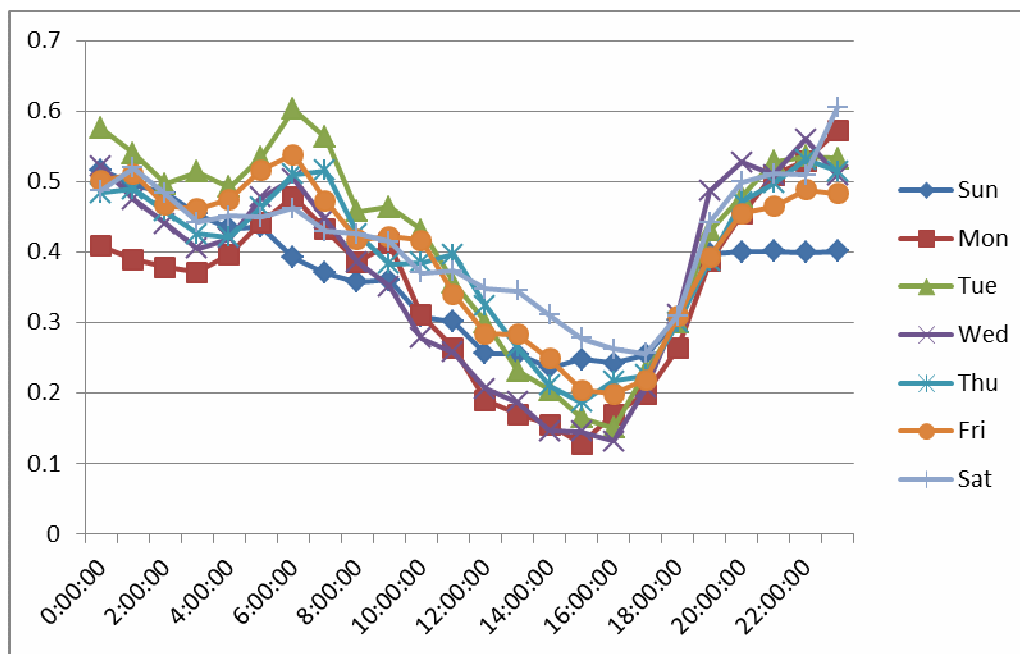


Figure B-37. Comparison of average hourly ethylbenzene concentrations at the Botanical Gardens, New York PAMS

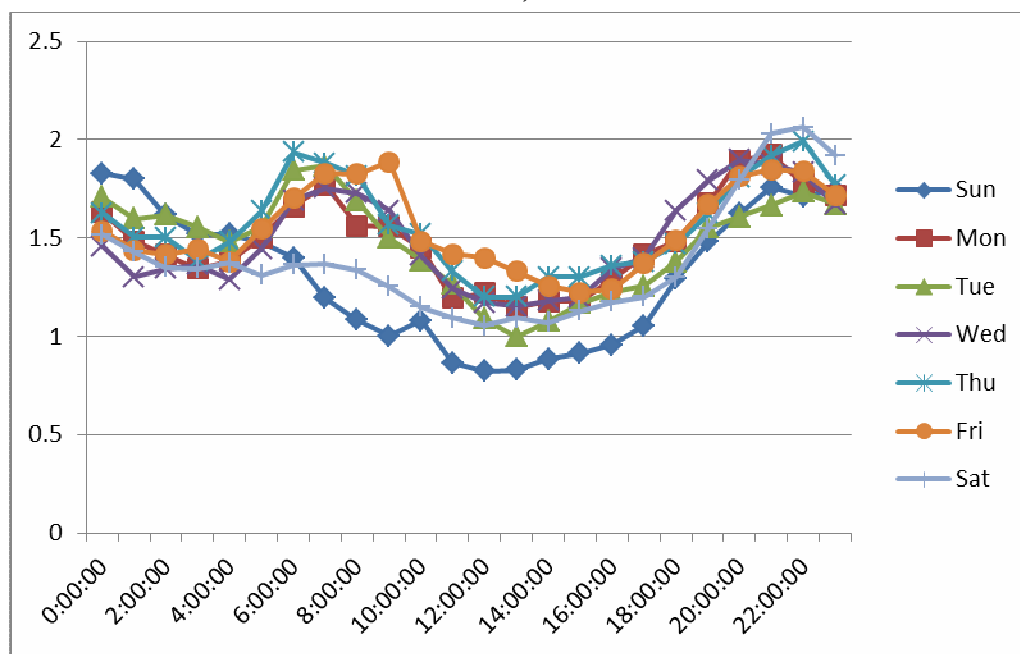


Figure B-38. Comparison of average hourly ethylbenzene concentrations at the Queens Community College, New York PAMS

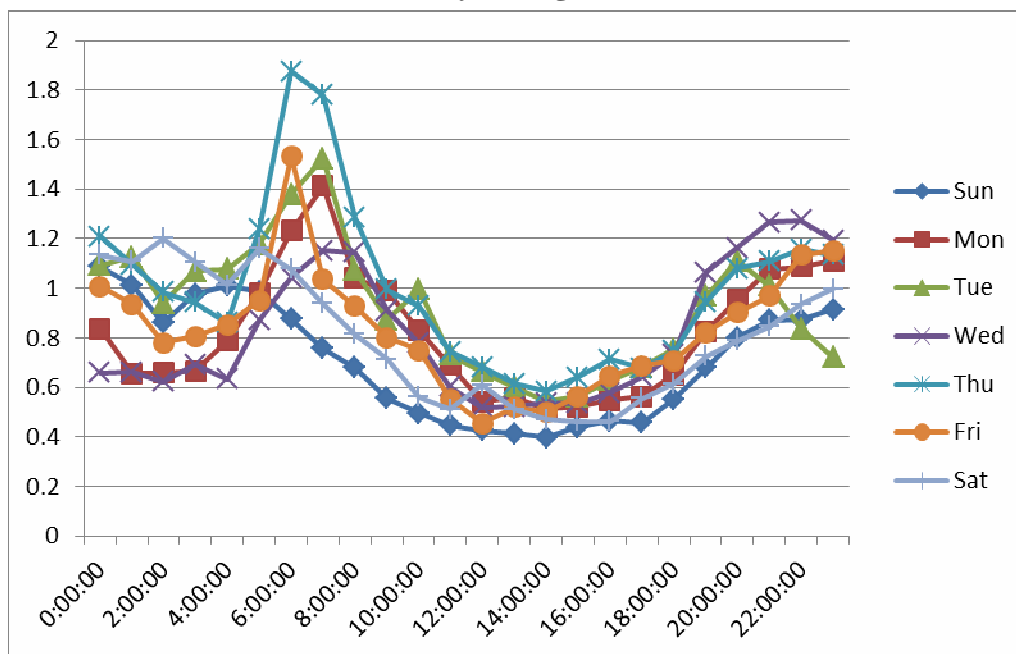


Figure B-39. Comparison of average hourly ethylbenzene concentrations at the East Lycoming, Pennsylvania PAMS

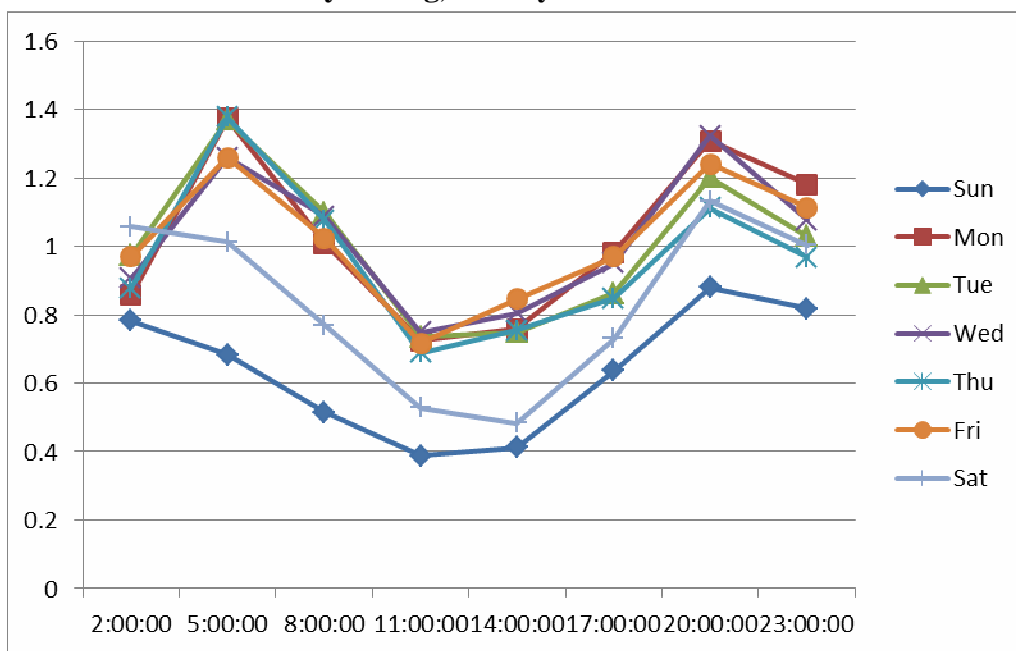
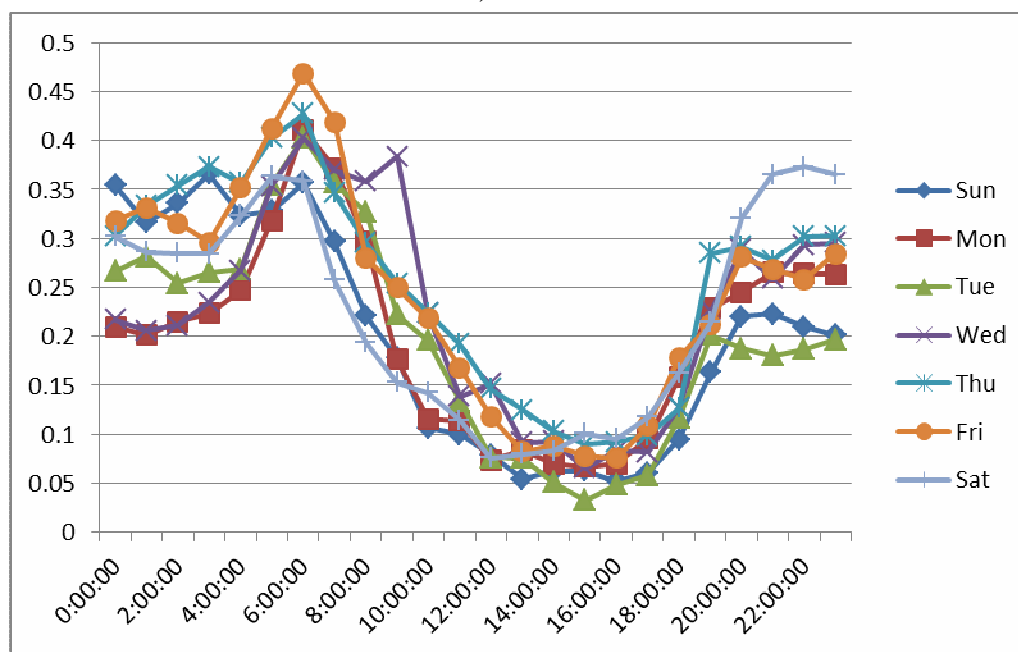
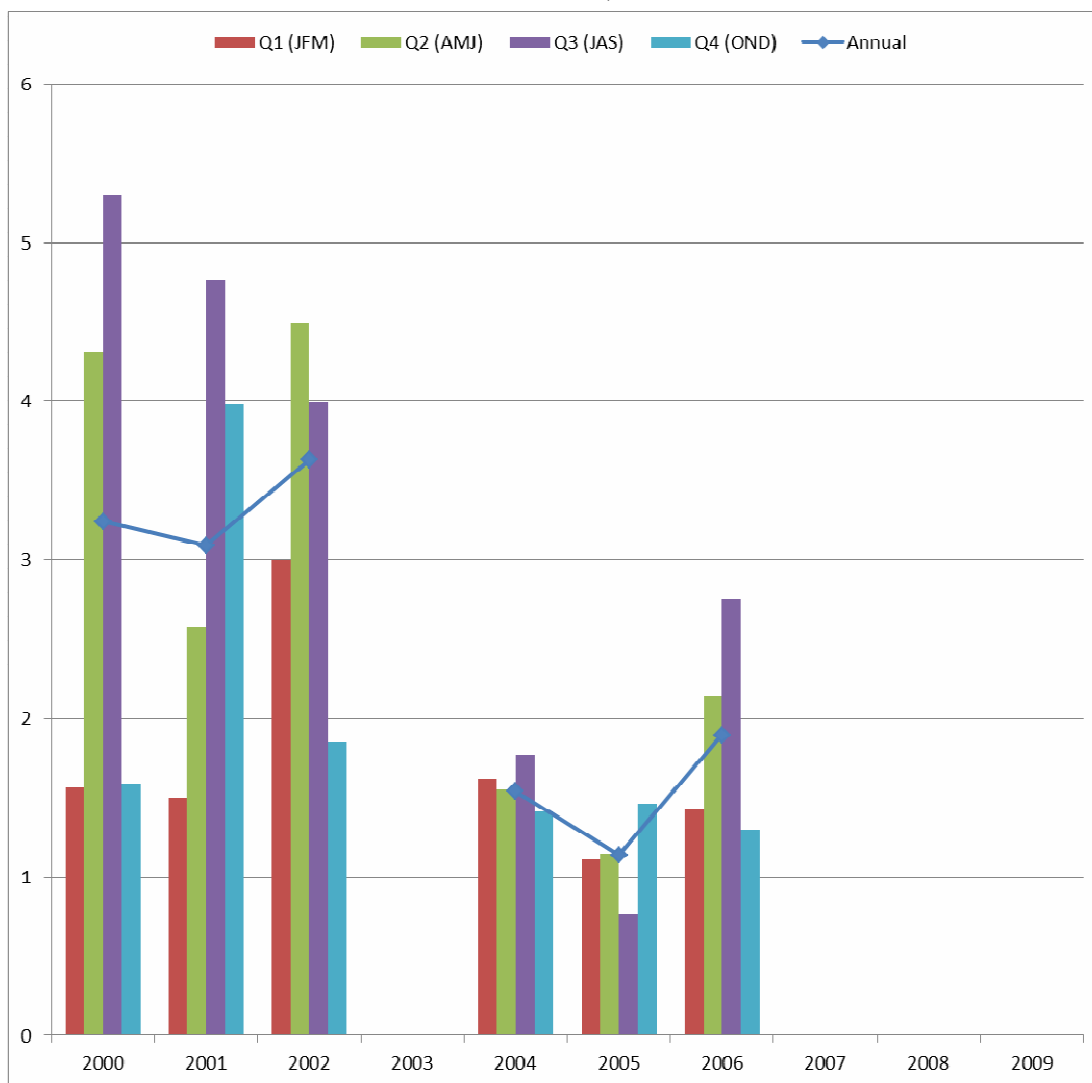


Figure B-40. Comparison of average hourly ethylbenzene concentrations at the Lums Pond, Delaware PAMS



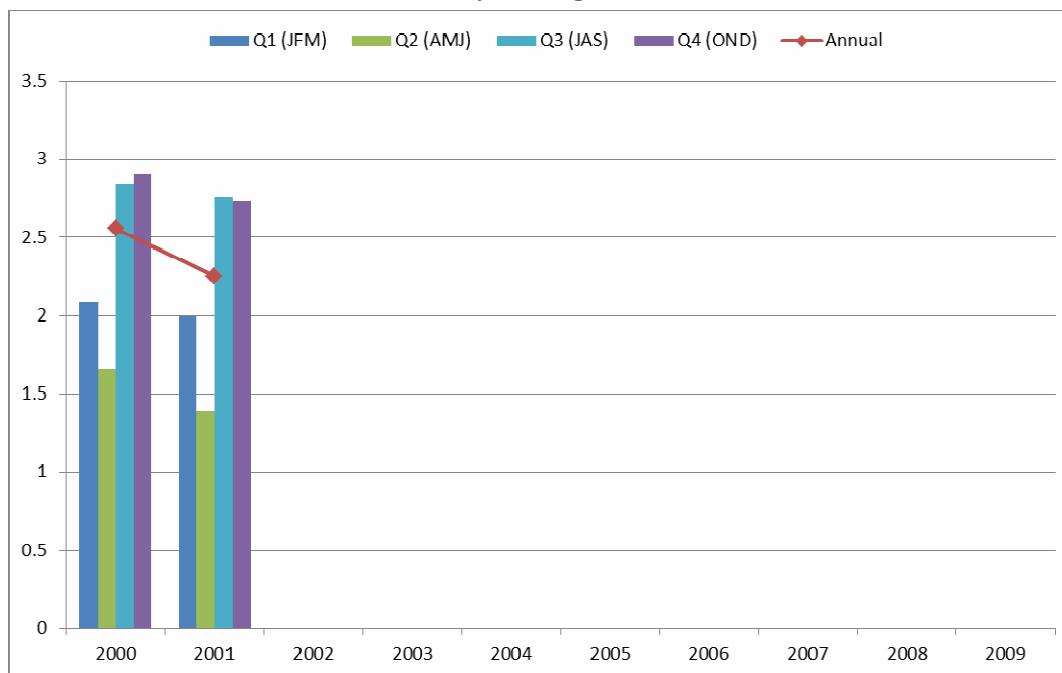
Formaldehyde

Figure B-41. Quarterly and annual average formaldehyde concentrations at Botanical Gardens, New York



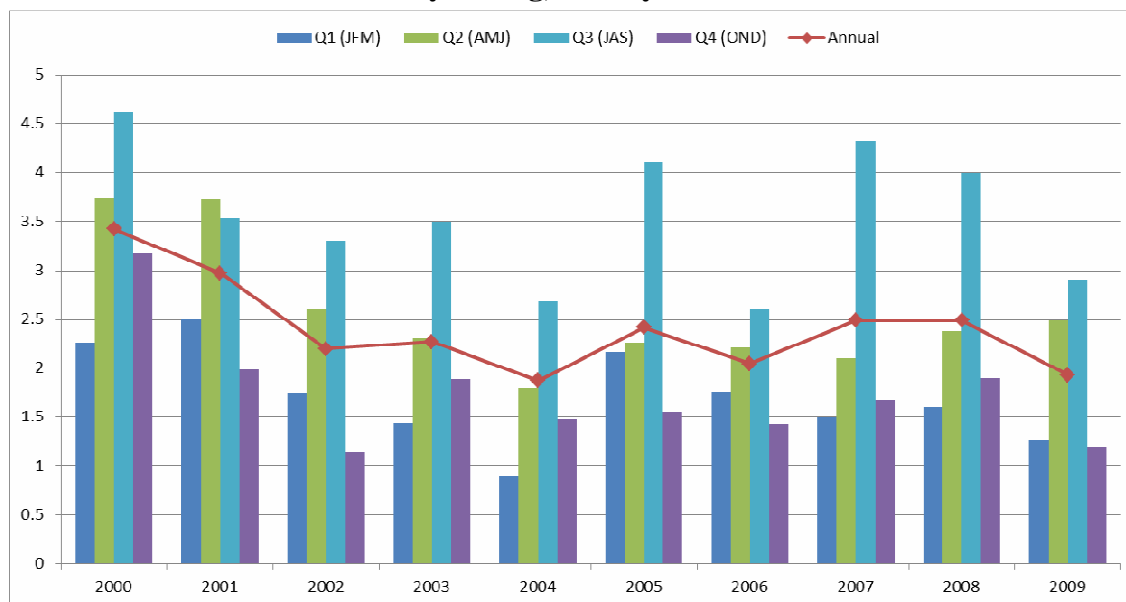
Note: Data were not available for 2003 or 2007 to present at this location. Data presented are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-42. Quarterly and annual average formaldehyde concentrations at Queens Community College, New York



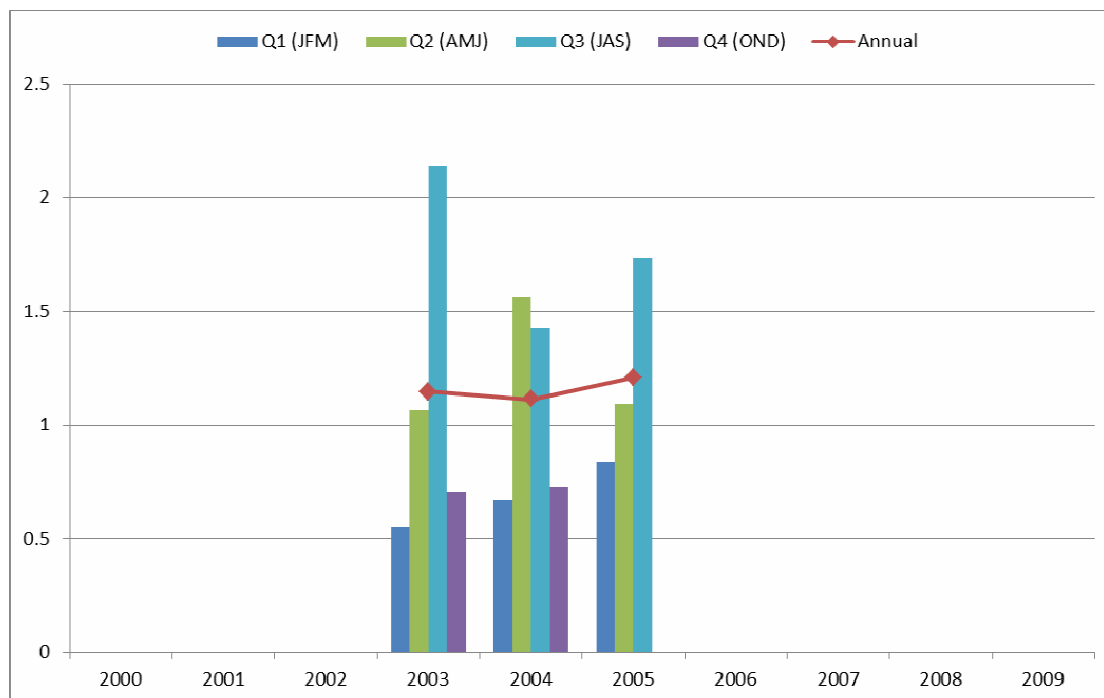
Note: Data were not available after 2001 for this location for formaldehyde. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-43. Quarterly and annual average formaldehyde concentrations at East Lycoming, Pennsylvania



Note: Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-44. Quarterly and annual average formaldehyde concentrations at Lums Pond, Delaware



Note: Data are only available between 2003 and Q3 2005. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-45. Comparison of average hourly formaldehyde concentrations at the Botanical Gardens, New York PAMS

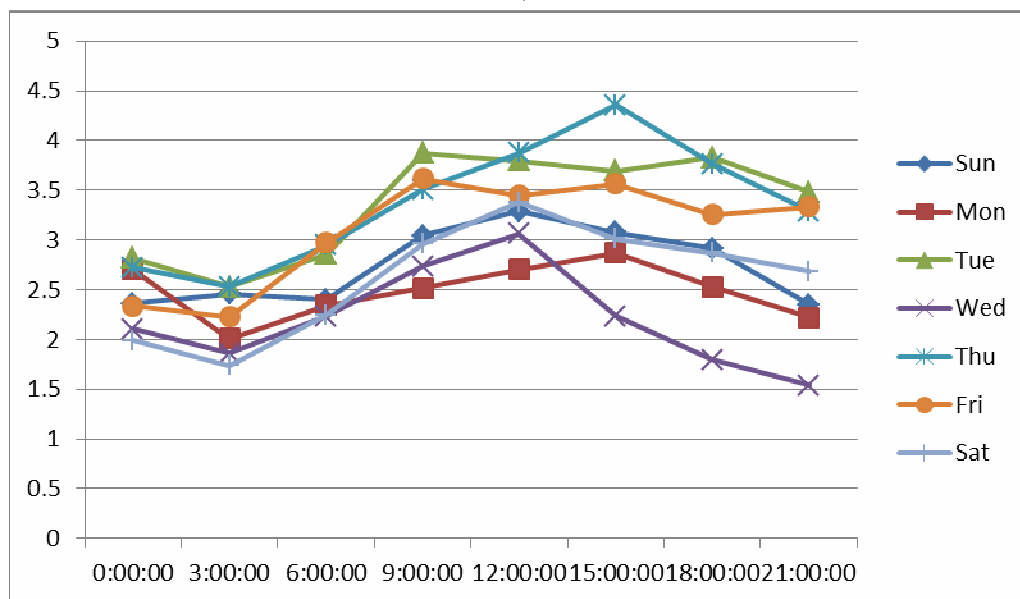


Figure B-46. Comparison of average hourly formaldehyde concentrations at the Queens Community College, New York PAMS

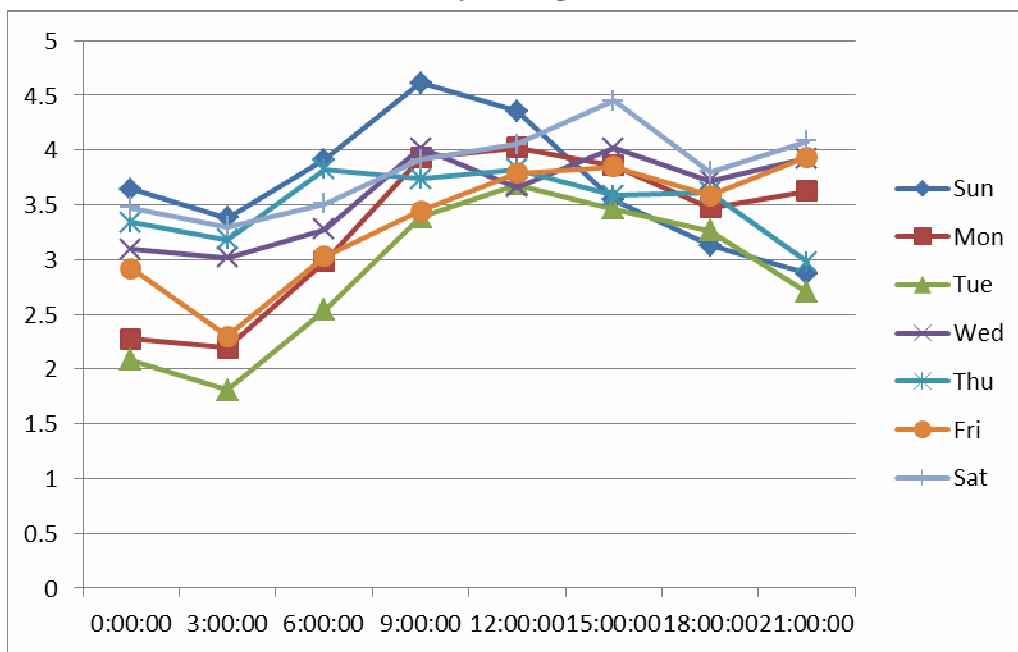
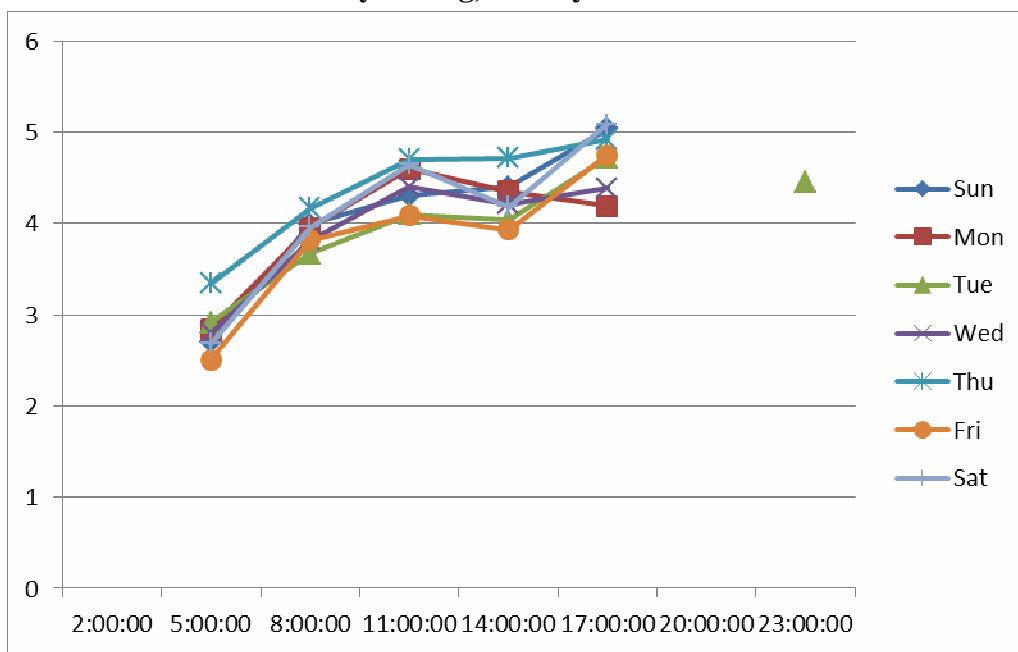
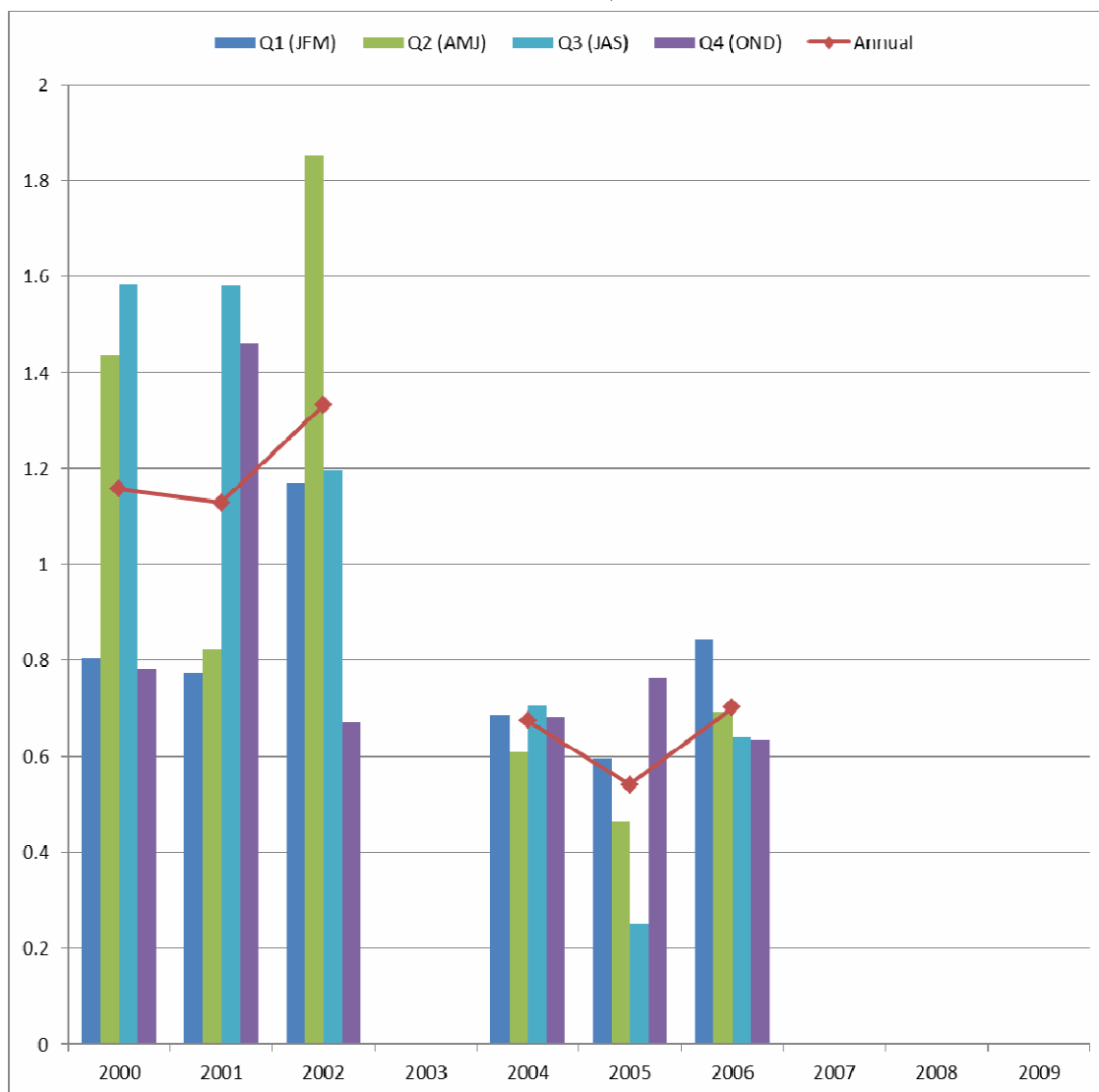


Figure B-47. Comparison of average hourly formaldehyde concentrations at the East Lycoming, Pennsylvania PAMS



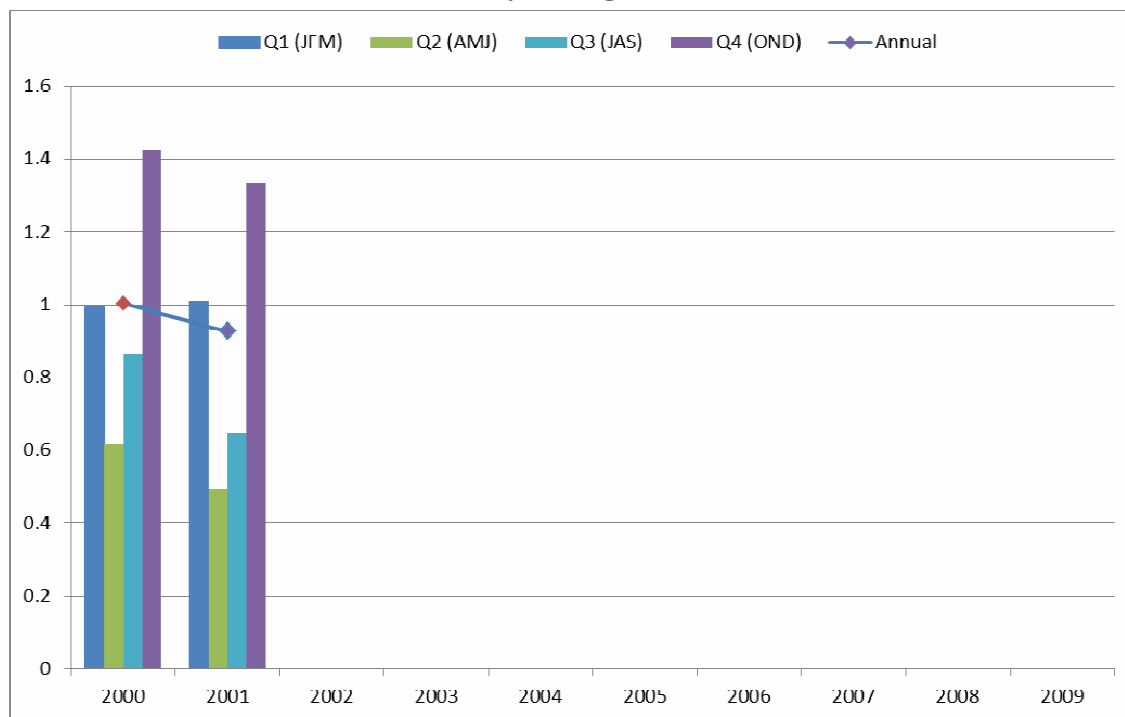
Acetaldehyde

Figure B-48. Quarterly and annual average acetaldehyde concentrations at Botanical Gardens, New York



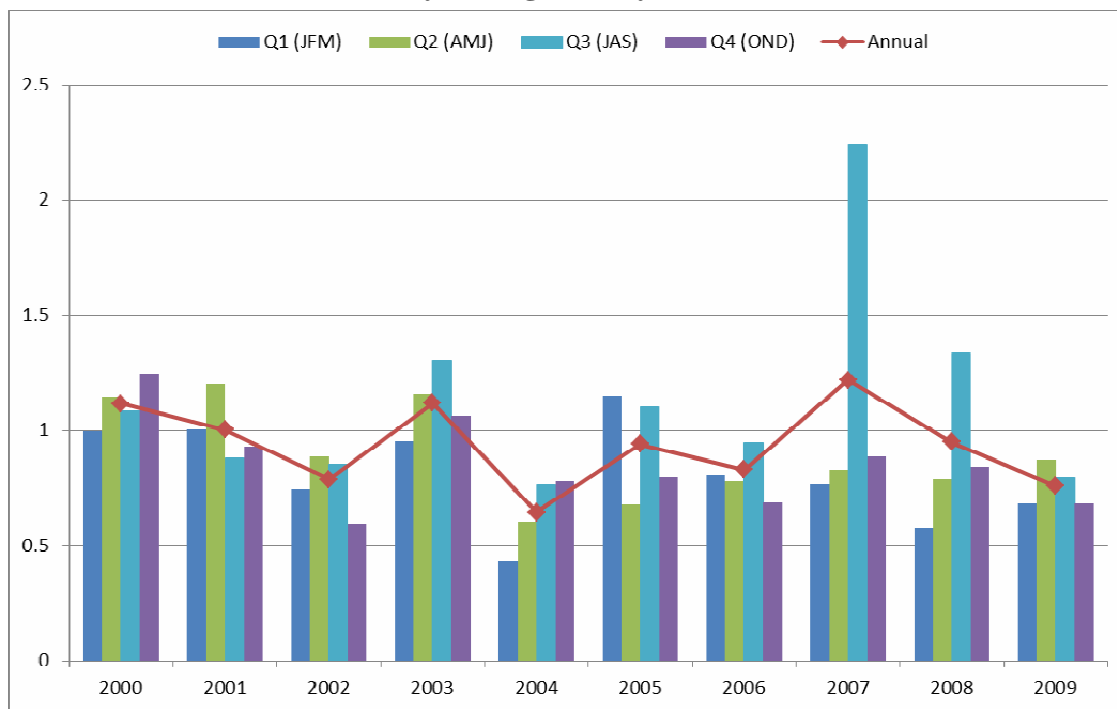
Note: Data were not available for 2003 or 2007 to present except for Q1 2006 at this location. Data presented are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-49. Quarterly and annual average acetaldehyde concentrations at Queens Community College, New York



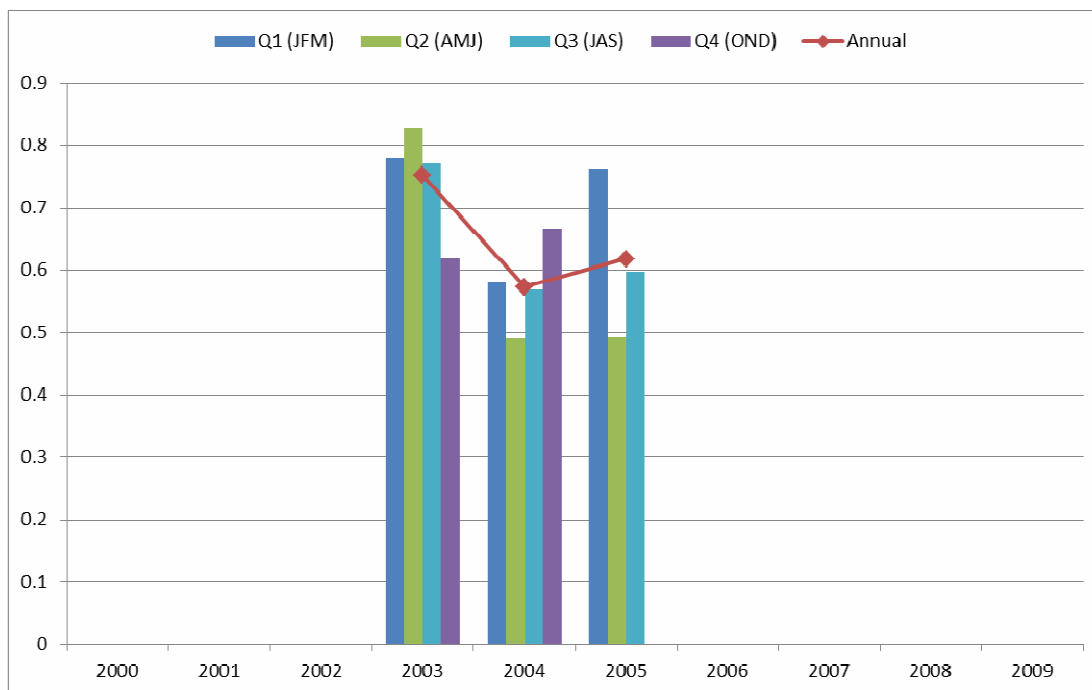
Note: Data were not available after 2001 for this location for acetaldehyde. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-50. Quarterly and annual average acetaldehyde concentrations at East Lycoming, Pennsylvania



Note: Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-51. Quarterly and annual average acetaldehyde concentrations at Lums Pond, Delaware



Note: Data were only available between 2003 and Q3 2005. Data are only from the 24-hour measurement site, not the collocated PAMS site. JFM = January, February, March; AMJ = April, May, June; JAS = July, August, September; OND = October, November, December.

Figure B-52. Comparison of average hourly acetaldehyde concentrations at the Botanical Gardens, New York PAMS

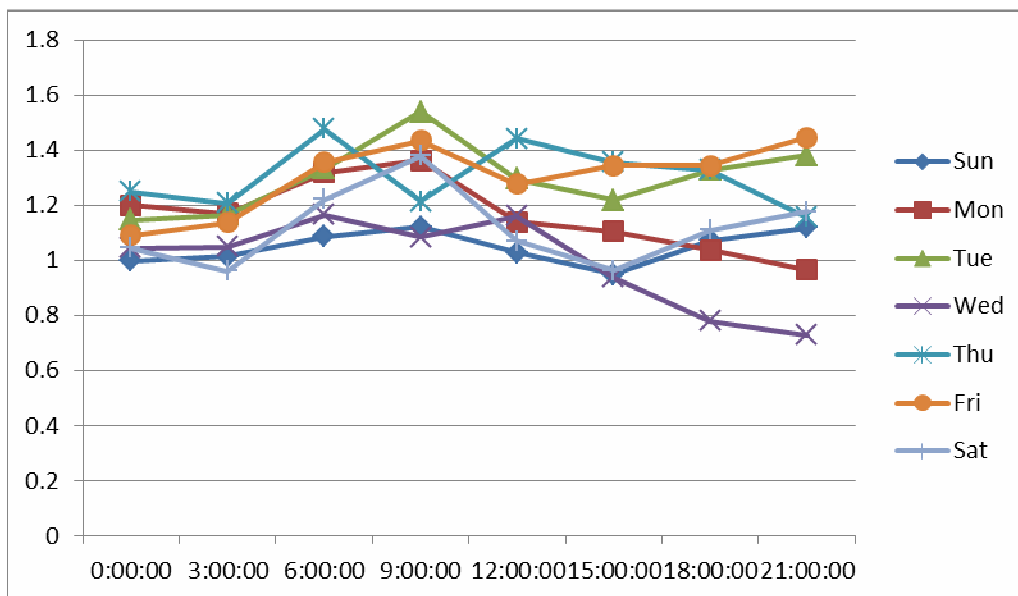


Figure B-53. Comparison of average hourly acetaldehyde concentrations at the Queens Community College, New York PAMS

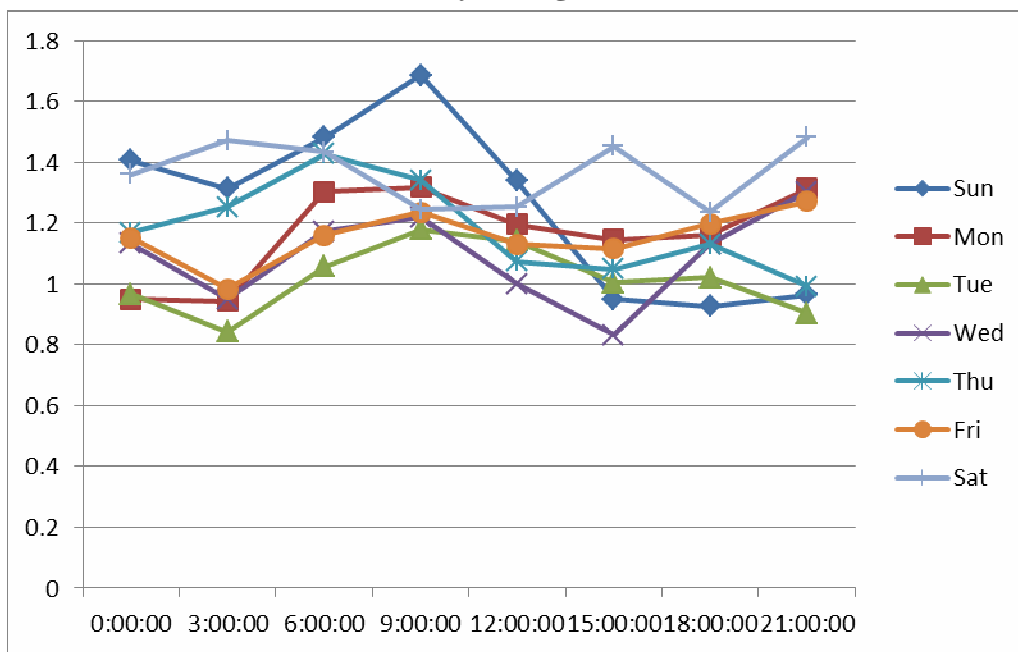


Figure B-54. Comparison of average hourly acetaldehyde concentrations at the East Lycoming, Pennsylvania PAMS

