

ASSESSMENT OF HEALTH BENEFITS FROM USING BIODIESEL AS A TRANSPORTATION FUEL AND RESIDENTIAL HEATING OIL



Clean Fuels
ALLIANCE AMERICA

Clean Fuels Alliance America

Prepared By:

Jeffrey Adkins, Principal Consultant
Jeremias Szust, Managing Consultant
Julia Ryan, Senior Consultant
Allan Daly, Senior Consultant
Sean Keane, Consultant

TRINITY CONSULTANTS
7919 Folsom Blvd
Suite 320
Sacramento, CA 95826
(916) 444-6666

March 2022
Rev. 2 April 2022

Trinity
Consultants 

TABLE OF CONTENTS

1. EXECUTIVE SUMMARY	1-13
1.1 Analysis Technique.....	1-13
1.2 Locations.....	1-13
1.3 Summary of Results	1-14
1.3.1 District of Columbia.....	1-14
1.3.2 Phoenix, Arizona.....	1-14
1.3.3 Chicago/Naperville, Illinois.....	1-15
1.3.4 Indianapolis, Indiana.....	1-15
1.3.5 Boston, Massachusetts.....	1-15
1.3.6 Detroit, Michigan.....	1-15
1.3.7 Minneapolis/St. Paul, Minnesota.....	1-16
1.3.8 Las Vegas, Nevada.....	1-16
1.3.9 Buffalo, New York.....	1-16
1.3.10 Port of New York/New Jersey, Port of Elizabeth	1-16
1.3.11 Charlotte, North Carolina.....	1-17
1.3.12 Cleveland/Akron/Canton, Ohio.....	1-17
1.3.13 Philadelphia-Reading-Camden (NJ), Pennsylvania.....	1-17
1.3.14 Houston, Texas.....	1-18
1.3.15 St. Louis, Missouri.....	1-18
1.4 Valuation of Health Benefit Results.....	1-18
1.5 Summary Tables	1-20
1.6 Importance of Health Benefits to Environmental Justice Communities.....	1-1
2. INTRODUCTION	2-1
2.1 Assessments of the Health Risk of Diesel Exhaust	2-1
2.1.1 U.S. EPA.....	2-1
2.1.2 California.....	2-1
3. OVERVIEW OF EXISTING DATA	3-1
3.1 U.S. EPA.....	3-1
3.1.1 2014 National Air Toxics Assessment (NATA).....	3-1
3.2 California.....	3-2
3.2.1 CalEnviroScreen.....	3-2
4. TOXICITY OF PETROLEUM DIESEL AND BIODIESEL EXHAUST	4-1
4.1 Toxicity of Petroleum-Derived Diesel Exhaust	4-1
4.1.1 U.S. EPA.....	4-1
4.1.2 CARB.....	4-1
4.2 Toxicity of Biodiesel Exhaust.....	4-2
5. HEALTH RISK ASSESSMENT METHODOLOGY	5-1
5.1 NATA Health Risk Assessment Methodology	5-1
5.2 NATA/HARP Hybrid Risk Assessment Methodology.....	5-1
5.3 Site-Specific Health Risk Assessments.....	5-1
5.4 Valuation of Health Risk Benefits.....	5-1
5.4.1 Geography, Incidence/Prevalence, and Population	5-2
5.4.2 Health Impacts.....	5-2

5.4.3	Valuation Functions	5-3
5.4.4	General Valuation Results	5-3

6. HEALTH RISK ASSESSMENT RESULTS

6-4

6.1	District of Columbia	6-4
6.1.1	NATA Health Risks.....	6-4
6.1.2	DC Site-Specific Health Risk Assessment.....	6-12
6.1.3	Valuation of Health Benefits.....	6-15
6.2	Phoenix, Arizona	6-16
6.2.1	NATA Risks	6-16
6.2.2	Phoenix Site-Specific Health Risk Assessment	6-24
6.2.3	Valuation of Health Benefits.....	6-27
6.3	Chicago/Naperville, Illinois.....	6-28
6.3.1	NATA Health Risks.....	6-28
6.3.2	Chicago/Naperville Site-Specific Health Risk Assessment	6-41
6.3.3	Valuation of Health Benefits.....	6-45
6.4	Indianapolis, Indiana	6-46
6.4.1	NATA Health Risks.....	6-46
6.4.2	Indianapolis Site-Specific Health Risk Assessment	6-54
6.4.3	Valuation of Health Benefits.....	6-57
6.5	Boston, Massachusetts.....	6-58
6.5.1	NATA Health Risks.....	6-58
6.5.2	Boston Site-Specific Health Risk Assessment	6-66
6.5.3	Valuation of Health Benefits.....	6-69
6.6	Detroit/Ann Arbor, Michigan.....	6-70
6.6.1	NATA Health Risks.....	6-70
6.6.2	Detroit Site-Specific Health Risk Assessment.....	6-90
6.6.3	Valuation of Health Benefits.....	6-93
6.7	Minneapolis/St. Paul, Minnesota.....	6-94
6.7.1	NATA Health Risks.....	6-94
6.7.2	Minneapolis/St. Paul Site-Specific Health Risk Assessment.....	6-102
6.7.3	Valuation of Health Benefits.....	6-105
6.8	Las Vegas, Nevada	6-106
6.8.1	NATA Health Risks.....	6-106
6.8.2	Las Vegas Site-Specific Health Risk Assessment	6-114
6.8.3	Valuation of Health Benefits.....	6-117
6.9	Buffalo, New York	6-118
6.9.1	NATA Health Risks.....	6-118
6.9.2	Toxicity of Heating Oil Combustion Compounds	6-119
6.9.3	Data Sources and Emissions Inventories.....	6-119
6.9.4	Models.....	6-120
6.9.5	NATA Modeling.....	6-121
6.9.6	Census-Specific Modeling.....	6-121
6.9.7	Buffalo, NY Health Risk Summary.....	6-122
6.9.8	Extrapolation of Risk Results to Other Similar Housing Areas	6-122
6.9.9	Valuation of Health Benefits.....	6-126
6.10	Port of Elizabeth, New York	6-127
6.10.1	NATA Health Risks.....	6-127
6.10.2	Port of Elizabeth Site-Specific Health Risk Assessment.....	6-141
6.10.3	Valuation of Health Benefits.....	6-145

6.11	Charlotte, North Carolina	6-146
	6.11.1 <i>NATA Health Risks</i>	6-146
	6.11.2 <i>Charlotte Site-Specific Health Risk Assessment</i>	6-154
	6.11.3 <i>Valuation of Health Benefits</i>	6-157
6.12	Cleveland, Ohio	6-158
	6.12.1 <i>NATA Health Risks</i>	6-158
	6.12.2 <i>Cleveland Site-Specific Health Risk Assessment</i>	6-166
	6.12.3 <i>Valuation of Health Benefits</i>	6-169
6.13	Philadelphia-Reading-Camden (NJ), Pennsylvania	6-170
	6.13.1 <i>NATA Health Risks</i>	6-170
	6.13.2 <i>Philadelphia Site-Specific Health Risk Assessment</i>	6-184
	6.13.3 <i>Valuation of Health Benefits</i>	6-187
6.14	Houston, Texas	6-188
	6.14.1 <i>NATA Health Risks</i>	6-188
	6.14.2 <i>Houston Site-Specific Health Risk Assessment</i>	6-196
	6.14.3 <i>Valuation of Health Benefits</i>	6-199
6.15	St. Louis, Missouri	6-200
	6.15.1 <i>NATA Health Risks</i>	6-200
	6.15.2 <i>St. Louis Site-Specific Health Risk Assessment</i>	6-208
	6.15.3 <i>Valuation of Health Benefits</i>	6-211

LIST OF FIGURES

Figure 6-1. DC Baseline NATA Total Cancer Risks	6-5
Figure 6-2. DC Baseline NATA DPM Cancer Risks	6-6
Figure 6-3. DC Reduced NATA DPM Cancer Risks	6-7
Figure 6-4. DC Baseline NATA DPM Concentrations	6-9
Figure 6-5. DC Baseline NATA/HARP DPM Hybrid Risks	6-10
Figure 6-6. DC Reduced NATA/HARP DPM Hybrid Risks	6-11
Figure 6-7. DC Baseline Health Risk Assessment Isopleths	6-13
Figure 6-8. DC Reduced Health Risk Assessment Risk Isopleths	6-14
Figure 6-9. Phoenix Baseline NATA Total Cancer Risks	6-17
Figure 6-10. Phoenix Baseline NATA DPM Cancer Risks	6-18
Figure 6-11. Phoenix Reduced NATA DPM Cancer Risks	6-19
Figure 6-12. Phoenix Baseline NATA DPM Concentrations	6-21
Figure 6-13. Phoenix Baseline NATA/HARP Hybrid Risks	6-22
Figure 6-14. Phoenix Reduced NATA/HARP Hybrid Risks	6-23
Figure 6-15. Phoenix Baseline Health Risk Assessment Isopleths	6-25
Figure 6-16. Phoenix Reduced Health Risk Assessment Risk Isopleths	6-26
Figure 6-17. North Chicago/Naperville Baseline NATA Total Cancer Risks	6-29
Figure 6-18. South Chicago/Naperville Baseline NATA Total Cancer Risks	6-30
Figure 6-19. North Chicago/Naperville Baseline NATA DPM Cancer Risks	6-31
Figure 6-20. South Chicago/Naperville Baseline NATA DPM Cancer Risks	6-32
Figure 6-21. North Chicago/Naperville Reduced NATA DPM Cancer Risks	6-33
Figure 6-22. South Chicago/Naperville Reduced NATA DPM Cancer Risks	6-34
Figure 6-23. North Chicago/Naperville Baseline NATA DPM Concentrations	6-36
Figure 6-24. South Chicago/Naperville Baseline NATA DPM Concentrations	6-37

Figure 6-25. North Chicago/Naperville Baseline NATA/HARP DPM Hybrid Risks	6-38
Figure 6-26. South Chicago/Naperville Baseline NATA/HARP DPM Hybrid Risks	6-39
Figure 6-27. North Chicago/Naperville Reduced NATA/HARP DPM Hybrid Risks	6-40
Figure 6-28. South Chicago/Naperville Reduced NATA/HARP DPM Hybrid Risks	6-41
Figure 6-29. Chicago/Naperville Baseline Health Risk Assessment Isopleths	6-43
Figure 6-30. Chicago/Naperville Reduced Health Risk Assessment Risk Isopleths	6-44
Figure 6-31. Indianapolis Baseline NATA Total Cancer Risk	6-47
Figure 6-32. Indianapolis Baseline NATA DPM Cancer Risks	6-48
Figure 6-33. Indianapolis Reduced NATA DPM Cancer Risks	6-49
Figure 6-34. Indianapolis Baseline NATA DPM Concentrations	6-51
Figure 6-35. Indianapolis Baseline NATA/HARP DPM Hybrid Risks	6-52
Figure 6-36. Indianapolis Reduced NATA/HARP DPM Hybrid Risks	6-53
Figure 6-37. Indianapolis Baseline Health Risk Assessment Isopleths	6-55
Figure 6-38. Indianapolis Reduced Health Risk Assessment Isopleths	6-56
Figure 6-39. Boston Baseline NATA Total Cancer Risks	6-59
Figure 6-40. Boston Baseline NATA DPM Cancer Risks	6-60
Figure 6-41. Boston Reduced NATA DPM Cancer Risks	6-61
Figure 6-42. Boston Baseline NATA DPM Concentrations	6-63
Figure 6-43. Boston Baseline NATA/HARP DPM Hybrid Risks	6-64
Figure 6-44. Boston Reduced NATA/HARP DPM Hybrid Risks	6-65
Figure 6-45. Boston Baseline Health Risk Assessment Isopleths	6-67
Figure 6-46. Boston Reduced Health Risk Assessment Isopleths	6-68
Figure 6-47. North Detroit Baseline NATA Total Cancer Risks	6-71
Figure 6-48. South Detroit Baseline NATA Total Cancer Risks	6-72
Figure 6-49. Ann Arbor Baseline NATA Total Cancer Risks	6-73
Figure 6-50. North Detroit Baseline NATA DPM Cancer Risks	6-74

Figure 6-51. South Detroit Baseline NATA DPM Cancer Risks	6-75
Figure 6-52. Ann Arbor Baseline NATA DPM Cancer Risks	6-76
Figure 6-53. North Detroit Reduced NATA DPM Cancer Risks	6-77
Figure 6-54. South Detroit Reduced NATA DPM Cancer Risks	6-78
Figure 6-55. Ann Arbor Reduced NATA DPM Cancer Risks	6-79
Figure 6-56. North Detroit Baseline NATA DPM Concentrations	6-81
Figure 6-57. South Detroit Baseline NATA DPM Concentrations	6-82
Figure 6-58. Ann Arbor Baseline NATA DPM Concentrations	6-83
Figure 6-59. North Detroit Baseline NATA/HARP DPM Hybrid Risks	6-84
Figure 6-60. South Detroit Baseline NATA/HARP DPM Hybrid Risks	6-85
Figure 6-61. Ann Arbor Baseline NATA/HARP DPM Hybrid Risks	6-86
Figure 6-62. North Detroit Reduced NATA/HARP DPM Hybrid Risks	6-87
Figure 6-63. South Detroit Reduced NATA/HARP DPM Hybrid Risks	6-88
Figure 6-64. Ann Arbor Reduced NATA/HARP DPM Hybrid Risks	6-89
Figure 6-65. Detroit/Ann Arbor Baseline Health Risk Assessment Isopleths	6-91
Figure 6-66. Detroit/Ann Arbor Reduced Health Risk Assessment Isopleths	6-92
Figure 6-67. Minneapolis/St. Paul Baseline NATA Total Cancer Risks	6-95
Figure 6-68. Minneapolis/St. Paul Baseline NATA DPM Cancer Risks	6-96
Figure 6-69. Minneapolis/St. Paul Reduced NATA DPM Cancer Risks	6-97
Figure 6-70. Minneapolis/St. Paul Baseline NATA DPM Concentrations	6-99
Figure 6-71. Minneapolis/St. Paul Baseline NATA/HARP DPM Hybrid Risks	6-100
Figure 6-72. Minneapolis/St. Paul Reduced NATA/HARP DPM Hybrid Risks	6-101
Figure 6-73. Minneapolis/St. Paul Baseline Health Risk Assessment Isopleths	6-103
Figure 6-74. Minneapolis/St. Paul Reduced Health Risk Assessment Isopleths	6-104
Figure 6-75. Las Vegas Baseline NATA Total Cancer Risks	6-107
Figure 6-76. Las Vegas Baseline NATA DPM Cancer Risks	6-108

Figure 6-77. Las Vegas Reduced NATA DPM Cancer Risks	6-109
Figure 6-78. Las Vegas Baseline NATA DPM Concentrations	6-111
Figure 6-79. Las Vegas Baseline NATA/HARP DPM Hybrid Risks	6-112
Figure 6-80. Las Vegas Reduced NATA/HARP DPM Hybrid Risks	6-113
Figure 6-81. Las Vegas Baseline Health Risk Assessment Isopleths	6-115
Figure 6-82. Las Vegas Reduced Health Risk Assessment Isopleths	6-116
Figure 6-83. Buffalo Cancer Risk Due to Home Heating Oil Combustion	6-124
Figure 6-84. Buffalo Reduced Cancer Risk Due to Biodiesel Heating Oil Combustion	6-125
Figure 6-85. Port of Elizabeth North Baseline NATA Total Cancer Risks	6-128
Figure 6-86. Port of Elizabeth South Baseline NATA Total Cancer Risks	6-129
Figure 6-87. Port of Elizabeth North Baseline NATA DPM Cancer Risks	6-130
Figure 6-88. Port of Elizabeth South Baseline NATA DPM Cancer Risks	6-131
Figure 6-89. Port of Elizabeth North Reduced NATA DPM Cancer Risks	6-132
Figure 6-90. Port of Elizabeth South Reduced NATA DPM Cancer Risks	6-133
Figure 6-91. Port of Elizabeth North Baseline NATA DPM Concentrations	6-135
Figure 6-92. Port of Elizabeth South Baseline NATA DPM Concentrations	6-136
Figure 6-93. Port of Elizabeth North Baseline NATA/HARP DPM Hybrid Risks	6-137
Figure 6-94. Port of Elizabeth South Baseline NATA/HARP DPM Hybrid Risks	6-138
Figure 6-95. Port of Elizabeth North Reduced NATA/HARP DPM Hybrid Risks	6-139
Figure 6-96. Port of Elizabeth South Reduced NATA/HARP DPM Hybrid Risks	6-140
Figure 6-97. Port of Elizabeth Baseline Health Risk Assessment Isopleths	6-142
Figure 6-98. Port of Elizabeth Reduced Health Risk Assessment Isopleths	6-144
Figure 6-99. Charlotte Baseline NATA Total Cancer Risks	6-147
Figure 6-100. Charlotte Baseline NATA DPM Cancer Risks	6-148
Figure 6-101. Charlotte Reduced NATA DPM Cancer Risks	6-149
Figure 6-102. Charlotte Baseline NATA DPM Concentrations	6-151

Figure 6-103. Charlotte Baseline NATA/HARP DPM Hybrid Risks	6-152
Figure 6-104. Charlotte Reduced NATA/HARP DPM Hybrid Risks	6-153
Figure 6-105. Charlotte Baseline Health Risk Assessment Isopleths	6-155
Figure 6-106. Charlotte Reduced Health Risk Assessment Isopleths	6-156
Figure 6-107. Cleveland Baseline NATA Total Cancer Risks	6-159
Figure 6-108. Cleveland Baseline NATA DPM Cancer Risks	6-160
Figure 6-109. Cleveland Reduced NATA DPM Cancer Risks	6-161
Figure 6-110. Cleveland Baseline NATA DPM Concentrations	6-163
Figure 6-111. Cleveland Baseline NATA/HARP DPM Hybrid Risks	6-164
Figure 6-112. Cleveland Reduced NATA/HARP DPM Hybrid Risks	6-165
Figure 6-113. Cleveland Baseline Health Risk Assessment Isopleths	6-167
Figure 6-114. Cleveland Reduced Health Risk Assessment Isopleths	6-168
Figure 6-115. S. Philadelphia Baseline NATA Total Cancer Risks	6-171
Figure 6-116. N. Philadelphia Baseline NATA Total Cancer Risks	6-172
Figure 6-117. S. Philadelphia Baseline NATA DPM Cancer Risks	6-173
Figure 6-118. N. Philadelphia Baseline NATA DPM Cancer Risks	6-174
Figure 6-119. S. Philadelphia Reduced NATA DPM Cancer Risks	6-175
Figure 6-120. N. Philadelphia Reduced NATA DPM Cancer Risks	6-176
Figure 6-121. S. Philadelphia Baseline NATA DPM Concentrations	6-178
Figure 6-122. N. Philadelphia Baseline NATA DPM Concentrations	6-179
Figure 6-123. S. Philadelphia Baseline NATA/HARP DPM Hybrid Risks	6-180
Figure 6-124. N. Philadelphia Baseline NATA/HARP DPM Hybrid Risks	6-181
Figure 6-125. S. Philadelphia Reduced NATA/HARP DPM Hybrid Risks	6-182
Figure 6-126. N. Philadelphia Reduced NATA/HARP DPM Hybrid Risks	6-183
Figure 6-127. Philadelphia Baseline Health Risk Assessment Isopleths	6-185
Figure 6-128. Philadelphia Reduced Health Risk Assessment Isopleths	6-186

Figure 6-129. Houston Baseline NATA Total Cancer Risks	6-189
Figure 6-130. Houston Baseline NATA DPM Cancer Risks	6-190
Figure 6-131. Houston Reduced NATA DPM Cancer Risks	6-191
Figure 6-132. Houston Baseline NATA DPM Concentrations	6-193
Figure 6-133. Houston Baseline NATA/HARP DPM Hybrid Risks	6-194
Figure 6-134. Houston Reduced NATA/HARP DPM Hybrid Risks	6-195
Figure 6-135. Houston Baseline Health Risk Assessment Isopleths	6-197
Figure 6-136. Houston Reduced Health Risk Assessment Isopleths	6-198
Figure 6-137. St. Louis Baseline NATA Total Cancer Risks	6-201
Figure 6-138. St. Louis Baseline NATA DPM Cancer Risks	6-202
Figure 6-139. St. Louis Reduced NATA DPM Cancer Risks	6-203
Figure 6-140. St. Louis Baseline NATA DPM Concentrations	6-205
Figure 6-141. St. Louis Baseline NATA/HARP DPM Hybrid Risks	6-206
Figure 6-142. St. Louis Reduced NATA/HARP DPM Hybrid Risks	6-207
Figure 6-143. St. Louis Baseline Health Risk Assessment Isopleths	6-209
Figure 6-144. St. Louis Reduced Health Risk Assessment Isopleths	6-210

LIST OF TABLES

Table 1-1. Summary of BenMAP Valuation Results	1-19
Table 1-2. NATA Total Cancer Risk and BenMAP Health Benefit Value Summary	1-20
Table 1-3. Summary of NATA Risks from DPM Sources	1-21
Table 1-4. NATA DPM Hybrid NATA/HARP Risk Calculation Summary	1-21
Table 1-5. Site-Specific Roadway HRA Results Summary	1-22
Table 4-1. Comparison of U.S. EPA and CARB Diesel Exhaust Toxicity Values	4-1
Table 6-1. DC Source Groups and Emission Rates	6-12
Table 6-2. DC Valuation of Reduced Incidence Benefits	6-15

Table 6-3. Phoenix Source Groups and Emission Rates	6-24
Table 6-4. Phoenix Valuation of Reduced Incidence Benefits	6-27
Table 6-5. Chicago/Naperville Source Groups and Emission Rates	6-42
Table 6-6. Chicago/Naperville Valuation of Reduced Incidence Benefits	6-45
Table 6-7. Indianapolis Source Groups and Emission Rates	6-54
Table 6-8. Indianapolis Valuation of Reduced Incidence Benefits	6-57
Table 6-9. Boston Source Groups and Emission Rates	6-66
Table 6-10. Boston Valuation of Reduced Incidence Benefits	6-69
Table 6-11. Detroit Source Groups and Emission Rates	6-90
Table 6-12. Detroit Valuation of Reduced Incidence Benefits	6-93
Table 6-13. Minneapolis/St. Paul Source Groups and Emission Rates	6-102
Table 6-14. Minneapolis/St. Paul Valuation of Reduced Incidence Benefits	6-105
Table 6-15. Las Vegas Source Groups and Emission Rates	6-114
Table 6-16. Las Vegas Valuation of Reduced Incidence Benefits	6-117
Table 6-17. Comparison of U.S. EPA and CARB Heating Oil Exhaust Toxicity Values	6-119
Table 6-18. Buffalo Census Tract 68 Modeling Parameters	6-120
Table 6-19. Buffalo NATA Risk Reduction for Census Tract 36029006800	6-121
Table 6-20. Buffalo Census-Specific Modeling	6-122
Table 6-21. Buffalo Valuation of Reduced Incidence Benefits	6-126
Table 6-22. Port of Elizabeth Source Groups and Emission Rates	6-141
Table 6-23. Port of Elizabeth Valuation of Reduced Incidence Benefits	6-145
Table 6-24. Charlotte Source Groups and Emission Rates	6-154
Table 6-25. Charlotte Valuation of Reduced Incidence Benefits	6-157
Table 6-26. Cleveland Source Groups and Emission Rates	6-166
Table 6-27. Cleveland Valuation of Reduced Incidence Benefits	6-169
Table 6-28. Philadelphia-Reading-Camden Source Groups and Emission Rates	6-184

Table 6-29. Philadelphia Valuation of Reduced Incidence Benefits	6-187
Table 6-30. Houston Source Groups and Emission Rates	6-196
Table 6-31. Houston Valuation of Reduced Incidence Benefits	6-199
Table 6-32. St. Louis Source Groups and Emission Rates	6-208
Table 6-33. St. Louis Valuation of Reduced Incidence Benefits	6-211

LIST OF ACRONYMS AND ABBREVIATIONS

AADT	Annual Average Daily Traffic
AERMOD	American Meteorological Society/Environmental Protection Agency Regulatory Model
B[a]anthracene	Benz[a]anthracene, a type of PAH
BenMAP-CE	Environmental Benefits Mapping and Analysis Program - Community Edition
CARB	California Air Resources Board
CalEnviroScreen	California Communities Environmental Health Screening Tool
CMAQ	Community Multiscale Air Quality Model
Cr(VI)	Hexavalent Chromium
CV	Contingent Valuation
D[a,h]anthracene	Dibenz[a,h]anthracene, a type of PAH
EMFAC	Emission Factor Model
EJSCREEN	Environmental Justice Screening and Mapping Tool
DPM	Diesel Particulate Matter
HAD	(Diesel) Hazard Assessment Document
HAP	Hazardous Air Pollutant
HARP	Hot Spots Analysis & Reporting Program
HRA	Health Risk Assessment
In[1,2,3-cd]pyr	Indeno[1,2,3-cd]pyrene, a type of PAH
LA	Los Angeles

LB	Long Beach
MEIR	Maximally Exposed Individual Resident
NAD	North American Datum
NATA	National Air Toxics Assessment
NEI	National Emissions Inventory
OEHHA	(California) Office of Environmental Health Hazard Assessment
NIOSH	National Institute for Occupational Safety and Health
PAH	Polycyclic Aromatic Hydrocarbon
PM _{2.5}	Particulate Matter with an Aerodynamic Diameter of ≤ 2.5 microns
PMI	Point of Maximum Impact
ppm	Parts Per Million
TSD	Technical Support Document
ULSD	Ultra-Low Sulfur Diesel
U.S. EPA	United States Environmental Protection Agency
UTM	Universal Transverse Mercator
DC	Wilmington, Carson, and West Long Beach
WTP	Willingness-to-Pay

This report assesses the health benefits of substituting biomass-based diesel in transportation-related sources currently fueled by conventional ultra-low sulfur diesel (ULSD or diesel fuel) at fourteen locations and as a replacement for home heating oil in one location throughout the United States. This study expands upon the Assessment of Health Benefits from Using Biodiesel as a Transportation Fuel and Residential Heating Oil completed by Trinity Consultants (Trinity) in 2021. The emission sources, data sources, models, and analytical techniques for each urbanized area were selected to provide the most comprehensive, robust, and transparent analysis possible within the schedule and budget limitations of the approved project. For all locations, Trinity has attempted to identify the communities believed to be most impacted by the emission sources modeled and has highlighted the benefits of biomass-based biodiesel to those specific communities to the degree possible.

1.1 Analysis Technique

The general analysis technique is a simplified, air toxic-based health risk assessment (HRA) of specific diesel fueled transportation-related sources and residential heating oil sources in the areas selected. The analyses do not attempt to replicate any existing HRA performed for a specific facility, correlate with monitored concentrations of specific pollutants, or quantify the full background health risk experienced in the area modeled. Rather, the analyses show the air toxic health risk benefits of fueling the modeled transportation-related and residential heating oil sources with biomass-based diesel compared to ULSD or distillate oil.

Because health risk is directly proportional to the established air pollutant toxicity values, the risk reduction percentage at any given location will be the same as the reduction in air pollutant toxicity from ULSD or distillate oil combustion compared to biomass-based diesel combustion. This analysis translates those changes in toxicity values into risk metrics, including reductions in cancer risk (per million people) and reduction in cancer burden.

USEPA's National Air Toxics Assessment (NATA) data was used to assess community-wide toxic pollutant concentrations and health risk values based on USEPA HRA procedures and pollutant toxicity values. This NATA concentration data was also used in the California Hot Spots Analysis & Reporting Program (HARP) HRA software to produce risk values based on California toxicity values and risk calculation procedures. Finally, separate site-specific HRAs were conducted to determine localized areas of concern near major roadways in each location analyzed for transportation fuel diesel particulate matter (DPM) impacts.

1.2 Locations

The following communities were assessed for health risk reductions from the use of biomass-based diesel in place of ULSD for transportation sources:

- > District of Columbia (DC)
- > Phoenix, Arizona
- > Chicago/Naperville, Illinois
- > Indianapolis, Indiana
- > Boston, Massachusetts
- > Metro Detroit, Michigan
- > Minneapolis/St. Paul, Minnesota
- > Las Vegas, Nevada

- > Buffalo, New York (biodiesel replacing home heating oil)
- > Port of New York/New Jersey, Port of Elizabeth, New York
- > Charlotte, North Carolina
- > Cleveland/Akron/Canton, Ohio
- > Philadelphia-Reading-Camden (NJ), Pennsylvania
- > Houston, Texas
- > St. Louis, Missouri

Each of the above locations was selected for specific reasons discussed in more detail within this report, but are generally those areas believed to be some of the most impacted by diesel emissions. For each location, multiple, and sometimes differing, data sources were available. This provided an opportunity to carry-out several types of analyses, which can be **broadly summarized as either “community-wide” or “source-specific.”** For all locations, both types of analyses were possible.

A secondary goal of this report is to compare and contrast the differences inherent in prior analyses, to examine the reasons for those differences, and to provide a bounded range of health risk benefits.

1.3 Summary of Results

1.3.1 District of Columbia

It is expected that the baseline cancer risk associated with diesel fuel usage in the DC area lies somewhere between 6 and 852 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel fuel to biomass-based diesel, the baseline cancer risk in the DC area is reduced to a value between 3 and 290 excess cancer cases per million residents.

The total cancer burden (cancer risk multiplied by affected population) for all census tracts in the DC study area is 293 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 100 with the use of biomass-based diesel fuel in place of traditional diesel.

The site-specific HRA for DC shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.2 Phoenix, Arizona

It is expected that the baseline cancer risk associated with diesel usage in the Phoenix area lies somewhere between 15 and 918 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel, the baseline cancer risk in Phoenix is reduced to a value between 6 and 308 excess cancer cases per million residents.

The total cancer burden for all census tracts in the Phoenix study area is 887 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 301 with the use of biomass-based diesel fuel in place of traditional diesel.

The site-specific HRA for Phoenix shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.3 Chicago/Naperville, Illinois

It is expected that the baseline cancer risk associated with diesel fuel usage in the Chicago/Naperville area lies somewhere between 9 and 1,812 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel, the baseline cancer risk in Chicago/Naperville is reduced to a value between 4 and 615 excess cancer cases per million residents.

The total cancer burden for all census tracts in the Chicago study area is 2,361 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 804 with the use of biomass-based diesel fuel in place of traditional ULSD.

The site-specific HRA for the Chicago area shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.4 Indianapolis, Indiana

It is expected that the baseline cancer risk associated with diesel fuel usage in the Indianapolis, Indiana area lies somewhere between 4 and 693 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel, the baseline cancer risk in Indianapolis is reduced to a value between 2 and 234 excess cancer cases per million residents.

The total cancer burden for all census tracts in the Indianapolis study area is 305 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 103 with the use of biomass-based diesel fuel in place of traditional diesel.

The site specific HRA for Indianapolis shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.5 Boston, Massachusetts

It is expected that the baseline cancer risk associated with diesel fuel usage in the Boston, Massachusetts area lies somewhere between 8 and 786 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel fuel, the baseline cancer risk in Boston is reduced to a value between 4 and 292 excess cancer cases per million residents.

The total cancer burden for all census tracts in the Boston study area is 364 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 129 with the use of biomass-based diesel fuel in place of traditional diesel.

The site specific HRA for Boston shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.6 Detroit, Michigan

It is expected that the baseline cancer risk associated with diesel fuel usage in the Detroit, Michigan area lies somewhere between 9 and 491 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel, the baseline cancer risk in Portland is reduced to a value between 4 and 180 excess cancer cases per million residents.

The total cancer burden for all census tracts in the Detroit study area is 1,007 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 351 with the use of biomass-based diesel fuel in place of traditional diesel.

The site specific HRA for Detroit shows that local risk maxima from a local project including regional highways and interstates is consistent with the evaluation of NATA data.

1.3.7 Minneapolis/St. Paul, Minnesota

It is expected that the baseline cancer risk associated with diesel fuel usage in the Minneapolis/St. Paul, Minnesota area lies somewhere between 6 and 585 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel, the baseline cancer risk in Minneapolis/St. Paul is reduced to a value between 3 and 192 excess cancer cases per million residents.

The total cancer burden for all census tracts in the Minneapolis/St. Paul study area is 376 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 127 with the use of biomass-based diesel fuel in place of traditional diesel.

The site specific HRA for Minneapolis/St. Paul shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.8 Las Vegas, Nevada

It is expected that the baseline cancer risk associated with diesel fuel usage in the Las Vegas, Nevada area lies somewhere between 13 and 947 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel, the baseline cancer risk in Las Vegas is reduced to a value between 6 and 321 excess cancer cases per million residents.

The total cancer burden for all census tracts in the Las Vegas study area is 612 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 209 with the use of biomass-based diesel fuel in place of traditional diesel.

The site specific HRA for Las Vegas shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.9 Buffalo, New York

It is expected that the baseline cancer risk associated with residential heating oil usage in the Buffalo, New York area lies somewhere between 0.3 and 6.0 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from residential distillate heating oil to biomass-based diesel, the baseline cancer risk in Buffalo is reduced to a value between 0.10 and 0.90 excess cancer cases per million residents.

The total cancer burden (cancer risk multiplied by affected population) for all census tracts in an 8-mile diameter circle centered over the center of Buffalo is 0.64 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 0.09 with the use of biomass-based diesel fuel for home heating oil combustion.

1.3.10 Port of New York/New Jersey, Port of Elizabeth

It is expected that the baseline cancer risk associated with diesel fuel usage in the Port of New York/New Jersey, Port of Elizabeth area lies somewhere between 17 and 2,227 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel,

the baseline cancer risk in Port of New York/New Jersey, Port of Elizabeth is reduced to a value between 7 and 890 excess cancer cases per million residents.

The total cancer burden (cancer risk multiplied by affected population) for all census tracts in the Port of New York/New Jersey, Port of Elizabeth study area is 3,911 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 1,394 with the use of biomass-based diesel fuel in place of traditional diesel.

The site specific HRA for the Port of New York/New Jersey, Port of Elizabeth shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.11 Charlotte, North Carolina

It is expected that the baseline cancer risk associated with diesel fuel usage in the Charlotte, North Carolina area lies somewhere between 7 and 584 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel, the baseline cancer risk in Charlotte is reduced to a value between 3 and 196 excess cancer cases per million residents.

The total cancer burden (cancer risk multiplied by affected population) for all census tracts in the Charlotte study area is 131 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 44 with the use of biomass-based diesel fuel in place of traditional diesel.

The site specific HRA for Charlotte shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.12 Cleveland/Akron/Canton, Ohio

It is expected that the baseline cancer risk associated with diesel fuel usage in the Cleveland/Akron/Canton, Ohio area lies somewhere between 5 and 509 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel, the baseline cancer risk in Cleveland/Akron/Canton is reduced to a value between 2 and 163 excess cancer cases per million residents.

The total cancer burden for all census tracts in the Cleveland/Akron/Canton study area is 208 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 70 with the use of biomass-based diesel fuel in place of traditional diesel.

The site specific HRA for Cleveland/Akron/Canton shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.13 Philadelphia-Reading-Camden (NJ), Pennsylvania

It is expected that the baseline cancer risk associated with diesel fuel usage in the Philadelphia area lies somewhere between 5 and 699 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel, the baseline cancer risk in the Philadelphia study area is reduced to a value between 2 and 227 excess cancer cases per million residents.

The total cancer burden for all census tracts in the Philadelphia study area is 695 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 247 with the use of biomass-based diesel fuel in place of traditional diesel.

The site specific HRA for Philadelphia shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.14 Houston, Texas

It is expected that the baseline cancer risk associated with diesel fuel usage in the Houston, Texas area lies somewhere between 8 and 887 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel, the baseline cancer risk in Houston is reduced to a value between 4 and 270 excess cancer cases per million residents.

The total cancer burden for all census tracts in the Houston study area is 998 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 340 with the use of biomass-based diesel fuel in place of traditional diesel.

The site specific HRA for Houston shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.3.15 St. Louis, Missouri

It is expected that the baseline cancer risk associated with diesel fuel usage in the St. Louis, Missouri area lies somewhere between 5 and 692 excess cancer cases per million residents over a 70-year timeline. Assuming a full transition from traditional diesel to biomass-based diesel, the baseline cancer risk in St. Louis is reduced to a value between 2 and 237 excess cancer cases per million residents.

The total cancer burden for all census tracts in the St. Louis study area is 630 assuming the higher end of the risk range, with an expected reduction in cancer burden to a value of approximately 221 with the use of biomass-based diesel fuel in place of traditional diesel.

The site specific HRA for St. Louis shows that local risk maxima from a local project including regional highways and interstates are consistent with the evaluation of NATA data.

1.4 Valuation of Health Benefit Results

The monetary valuation of the health benefits associated with using biodiesel as a transportation fuel or as residential heating oil was evaluated for each community. The benefits are based on reductions of ambient PM_{2.5} concentrations as discussed within this report, coupled with the incidence/prevalence rates and population of the area. These health benefit valuations **were calculated using U.S. EPA's BenMAP program, using the US.EPA's configuration files (dated April 19, 2021)**, named as follows:

- > *U.S. EPA approach for quantifying and valuing PM effects (zip)*; and,
- > *U.S. EPA approach for quantifying and valuing ozone effects (zip)*.¹

¹ Available at: [BenMAP Community Edition | US EPA](#), Accessed 3/28/2022.

The above configuration files represent the methodology described in *U.S. EPA's Technical Support Document: Estimating PM2.5- and Ozone-Attributable Health Benefits*.² The overall benefit rates for each health endpoint are shown in Error! Reference source not found..

Table 1-1. Summary of BenMAP Valuation Results

Endpoint	BenMAP Valuation Used in this Report ^a
Acute Myocardial Infarction Nonfatal	\$32,023 per incidence
Asthma Symptoms - Albuterol Use	\$0 per incidence ^b
Emergency Room (ER) Visits - All Cardiac Outcomes	\$1,161 per ER visit
Emergency Room (ER) visits - Respiratory	\$875 per ER visit
Hospital Admissions (HA) - All Respiratory	\$17,721 per HA
Hospital Admissions (HA) - Alzheimer's Disease	\$12,216 per HA
Hospital Admissions (HA) - Cardio- Cerebro- and Peripheral Vascular Disease	\$15,584 per HA
Hospital Admissions (HA) - Parkinson's Disease	\$12,921 per HA
Hospital Admissions (HA) - Respiratory-2	\$0 per HA
Hospital Admissions (HA) - Respiratory-2 – All Respiratory	\$0 per HA
Incidence - Asthma	\$44,657 per incidence
Incidence - Hay Fever/Rhinitis	\$600 per incidence
Incidence - Lung Cancer	\$12,669 per incidence
Incidence - Out of Hospital Cardiac Arrest	\$35,753 per incidence
Incidence - Stroke	\$33,962 per incidence
Minor Restricted Activity Days	\$70 per day
Mortality - All Cause	\$7,807,449 per death
Work Loss Days	\$190 per day

a. Average valuation used in the report. Valuations vary minimally based on location.

b. Actual value is \$0.35 per incidence, resulting in benefit values above zero in the location benefit valuation tables.

² U.S. EPA, Technical Support Document (TSD) for the Final Revised Cross-State Air Pollution Rule Update for the 2008 Ozone Season NAAQS, Docket ID No. EPA-HQ-OAR-2020-0272, Available at: [estimating_pm2.5- and ozone-attributable_health_benefits_tsd_march_2021.pdf \(epa.gov\)](https://www.epa.gov/estimating-pm2.5-and-ozone-attributable-health-benefits-tsd-march-2021.pdf), Accessed 3/28/2022.

1.5 Summary Tables

The following tables summarize the cancer risk data and health benefit values from the USEPA’s NATA database, the California HARP model results, the localized roadway HRA results, and the USEPA’s BenMAP model.

Table 1-2. NATA Total Cancer Risk and BenMAP Health Benefit Value Summary

Location	NATA Max Baseline Total Cancer Risk (10 ⁻⁶)	NATA Total Cancer Burden	BenMAP Health Benefit Value (million \$)
District of Columbia	46	20	262
Phoenix	59	89	354
Chicago/Naperville	89	155	677
Indianapolis	35	21	117
Boston	40	28	168
Detroit/Ann Arbor	47	98	267
Minneapolis/St. Paul	40	36	156
Las Vegas	51	50	202
Buffalo (Home Heating Oil)	0.34	N/A	101
Port of Elizabeth	57	164	1,433
Charlotte	53	15	70
Cleveland/Akron/Canton	57	16	112
Philadelphia/Reading/Camden	41	61	366
Houston	224	96	302
St. Louis	50	56	212

Table 1-3. Summary of NATA Risks from DPM Sources

Location	NATA Max Baseline DPM Cancer Risk (10 ⁻⁶)	NATA DPM Cancer Burden	Reduced NATA DPM Cancer Risk (10 ⁻⁶)	Reduced NATA DPM Cancer Burden
District of Columbia	6	2	3	1
Phoenix	15	9	6	4
Chicago/Naperville	9	18	4	7
Indianapolis	4	2	2	1
Boston	8	4	4	2
Detroit/Ann Arbor	9	9	4	4
Minneapolis/St. Paul	6	4	3	2
Las Vegas	13	7	6	3
Buffalo (Home Heating Oil)	0.3	<1	0.1	<1
Port of Elizabeth	17	33	7	14
Charlotte	7	1	3	<1
Cleveland/Akron/Canton	5	2	2	1
Philadelphia/Reading/Camden	5	6	2	3
Houston	8	7	4	3
St. Louis	5	4	2	2

Table 1-4. NATA DPM Hybrid NATA/HARP Risk Calculation Summary

Location	NATA/HARP Max Baseline DPM Cancer Risk (10 ⁻⁶)	NATA/HARP DPM Cancer Burden	NATA/HARP Reduced DPM Max Cancer Risk (10 ⁻⁶)	NATA/HARP Reduced DPM Cancer Burden
District of Columbia	852	293	290	100
Phoenix	918	887	308	301
Chicago/Naperville	1,812	2,361	615	804
Indianapolis	693	305	234	103
Boston	786	364	292	129
Detroit/Ann Arbor	491	1,007	180	351
Minneapolis/St. Paul	585	376	192	127
Las Vegas	947	612	321	209
Buffalo (Home Heating Oil)	6	<1	1	<1
Port of Elizabeth	2,227	3,911	890	1,394
Charlotte	584	131	196	44
Cleveland/Akron/Canton	509	208	163	70
Philadelphia/Reading/Camden	699	695	227	247
Houston	887	998	270	340
St. Louis	692	630	237	221

Table 1-5. Site-Specific Roadway HRA Results Summary

Location	Roadway HRA Max Baseline PMI (10 ⁻⁶)	Roadway HRA Max Baseline MEIR (10 ⁻⁶)	Roadway HRA Reduced PMI (10 ⁻⁶)	Roadway HRA Reduced MEIR (10 ⁻⁶)
District of Columbia	793	640	320	258
Phoenix	1,449	1,423	584	573
Chicago/Naperville	1,245	1,245	502	502
Indianapolis	790	590	318	238
Boston	480	379	194	153
Detroit/Ann Arbor	684	470	276	189
Minneapolis/St. Paul	981	684	395	276
Las Vegas	1,746	1,194	703	481
Buffalo (Home Heating Oil)	N/A	N/A	N/A	N/A
Port of Elizabeth	768	670	309	270
Charlotte	1,283	946	517	381
Cleveland/Akron/Canton	835	771	336	311
Philadelphia/Reading/Camden	617	535	382	216
Houston	1,446	1,124	583	453
St. Louis	1,446	1,446	583	583

1.6 Importance of Health Benefits to Environmental Justice Communities

The U.S. EPA defines environmental Justice (EJ) as **“the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies.”**³ One element of EJ is **“The same degree of protection from environmental and health hazards.”**⁴

As shown in the figures within this report, the health impacts of diesel emissions are disparately high in areas in close proximity to ports, railyards, distribution centers, freeways, and major roadways. These areas also frequently correspond to areas exhibiting elevated incidence rates of EJ metrics, such as:

- Asthma
- Low Birth Rate
- Cardiovascular Disease
- Low Education
- Linguistic Isolation
- Poverty
- Unemployment
- Housing Burden

These areas are also those with concentrated diesel engine activity of all categories (on-road, off-road, locomotives, and marine). Additionally, the **fraction of the diesel engine “population” in these areas tends to** be skewed toward conventional diesel engines, rather than new technology diesel engines equipped with particulate matter control systems and selective catalytic reduction (SCR) systems (to control emissions of nitrogen oxides). As such, EJ communities tend to be exposed to (i) higher concentrations of diesel exhaust in general, and (ii) higher concentrations of exhaust emitted by older diesel engines.

The thrust of this study is to demonstrate the benefits of the substitution of biodiesel for conventional diesel. The communities selected for this study were those identified to experience the highest emission rates, the highest ambient concentrations, and the highest risk levels due to diesel exhaust.

As described within this report, these benefits are credited to older technology diesel engines, which, in general, are those not meeting 2010 on-highway certification standards (for on-road engines), and those not meeting Tier 4 final certification standards (for nonroad engines). These benefits of biodiesel will therefore accrue to a much greater degree within EJ communities.

³ See: [Environmental Justice | US EPA](#)

⁴ Ibid.

2.1 Assessments of the Health Risk of Diesel Exhaust

Diesel exhaust has been identified as an air toxic by the U.S. EPA and the California Air Resources Board (CARB). The following sections provide an overview of the diesel exhaust toxic review process performed by these agencies.

2.1.1 U.S. EPA

With respect to this analysis, U.S. EPA's current stance on the toxicity of diesel particulate matter (DPM) as a carcinogen is outlined in the NATA Technical Support Document (TSD), which states the following about DPM in Section 5.4.6.

Diesel PM (DPM) mass (expressed as $\mu\text{g DPM}/\text{m}^3$) has historically been used as a surrogate measure of exposure for whole diesel exhaust. Although uncertainty exists as to whether DPM is the most appropriate parameter to correlate with human health effects, it is considered a reasonable choice until more definitive information about the mechanisms of toxicity or mode(s) of action of diesel exhaust becomes available.

In U.S. **EPA's 2002 Diesel Health Assessment Document (Diesel HAD), exposure to diesel exhaust was** classified as likely to be carcinogenic to humans by inhalation from environmental exposures, in accordance with the revised draft 1996/1999 U.S. EPA cancer guidelines. Several other agencies (National Institute for Occupational Safety and Health, the International Agency for Research on Cancer, the World Health Organization, California EPA and the U.S. Department of Health and Human Services) had made similar hazard classifications prior to 2002. U.S. EPA also concluded in the 2002 Diesel HAD that it was impossible to calculate a cancer unit risk for diesel exhaust due to limitations in the exposure data for the occupational groups or the absence of a dose-response relationship.

In the absence of a cancer unit risk, the Diesel HAD sought to provide additional insight into the significance of the diesel exhaust cancer hazard by estimating possible ranges of risk that might be present in the population. An exploratory analysis was used to characterize a possible risk range, and found that environmental risks from diesel exhaust exposure could plausibly range from a low of 10^{-5} to as high of 10^{-3} for long-term exposures. Because of uncertainties, the analysis acknowledged that "the risks could be lower than 10^{-5} , **and a zero risk from diesel exhaust exposure was not ruled out.**"

Additionally, the Diesel HAD **states in Section 1.9.1 that** "[t]he results do not include exposures and risk from all compounds." Of note, the assessment does not quantify cancer risk from diesel PM, although EPA has concluded that "the general population is exposed to levels close to or overlapping with levels that have **been linked to increased cancer risk in epidemiology studies.**"

The NATA study instead quantifies the carcinogenic effects of DPM using the toxicity factors and exposure levels comprising diesel exhaust. For this reason, this analysis will utilize the NATA determined DPM excess cancer risk values as a low-end estimate of baseline and reduced cancer risks.

2.1.2 California

The California Air Resources Board (CARB) has **designated** "particulate emissions from diesel-fueled engines," **commonly referred to as** diesel particulate matter or "DPM," as a Toxic Air Contaminant. This

determination is based primarily on evidence from occupational studies that show a link between exposure to DPM and lung cancer induction, as well as death from lung cancer.⁵ According to CARB, DPM air toxics are related to 70% of known cancer risks California and are estimated to increase statewide cancer risk by 520 cancers per million residents exposed over a lifetime.

Additionally, the Office of Environmental Health Hazard Assessment (OEHHA) cancer unit risk factor is approximately 100 times greater than the cumulative cancer risks estimated by U.S. EPA for individual diesel exhaust components. As such, this analysis will generate excess cancer risk values based on the OEHHA DPM values using **CARB's Hot Spots Analysis & Reporting Program (HARP) software as a high-end estimate** of baseline and reduced cancer risks.

⁵ <https://ww2.arb.ca.gov/resources/overview-diesel-exhaust-and-health>

3. OVERVIEW OF EXISTING DATA

3.1 U.S. EPA

3.1.1 2014 National Air Toxics Assessment (NATA)

U.S. EPA has generated an **interactive tool, EJSCREEN, that allows the EPA to “better meet the Agency’s responsibilities related to the protection of public health and the environment”**. EJSCREEN is an environmental justice mapping and screening tool that includes information such as census tract DPM concentrations and risk values. The basis for those risk values is the NATA.

According to the NATA TSD, “[t]he NATA is the U.S. EPA’s ongoing thorough evaluation of air toxics across the United States.” EPA developed NATA as a state-of-the-science tool to inform both national and localized efforts to collect air toxics information, characterize emissions and help prioritize pollutants and areas of interest for further study to gain a better understanding of risks. The goal of NATA is to identify those air toxics which are of greatest potential concern in terms of contribution to population risk. Ambient and exposure concentrations and estimates of risk and hazard for air toxics in each state are typically generated at the census tract level.

U.S. EPA determines county-wide health risks from DPM by determining the health risks associated with individual component risks as part of the National Emission Inventory (NEI) dataset, which is subsequently evaluated in the NATA. The NATA also evaluates total DPM concentrations on a census tract level, even though that information is not used to generate risk values directly. Those DPM concentrations can be **utilized in CARB’s HARP program to determine overall DPM cancer risk values using OEHHA derived** risk factors.

The types of sources that contribute to modeled DPM concentrations include the following sources identified in the NATA:

- > On-road sources,
- > Nonroad sources,
- > Point-airport-ground support equipment,
- > Point-locomotives,
- > Nonpoint locomotives, and
- > All PM from Diesel or residual-oil-fueled nonpoint commercial marine vessels.

It should be noted that while DPM emissions are not directly recorded for other nonpoint emission sources, such as fuel combustion of distillate fuel oil, the component Hazardous Air Pollutant (HAP) emissions of those sources are reported in the NEI and analyzed in the NATA⁶. Those sources are not included in this analysis as only the directly calculated DPM concentrations are utilized, and the census-by-census analysis using NATA data is not a full picture of census-specific DPM emissions or concentrations. Therefore, these analyses using DPM concentrations are likely understating the potential baseline and reduced cancer risks estimated herein.

The NATA TSD states that DPM emissions from 2014 NEI sources were modeled using a hybrid approach with the Community Multiscale Air Quality (CMAQ) and AERMOD models for the 52 most prevalent and high-

⁶ This information was used to conduct the fuel heating oil analyses

risk toxics, including DPM. Coarse, region-wide impacts were determined on a county level using 12-kilometer grids in the CMAQ model. AERMOD was utilized to generate near-field concentrations using gridded receptors (1 km in highly populated areas [>1 million population], 4 km in other areas), census block centroid receptors, and monitoring site receptors. These results were then weighted according to grid cell averages to determine census block and tract exposures for the 52 toxics. All other toxics were modeled directly using AERMOD.

Section 5 describes how the NATA data was utilized to determine EPA derived DPM baseline and reduced risks, and OEHHA derived DPM baseline and reduced risks.

3.2 California

3.2.1 CalEnviroScreen

CARB has also generated an environmental justice tool known as the California Communities Environmental Health Screening Tool (CalEnviroScreen). CalEnviroScreen does not utilize NATA data for its environmental justice tool, and it provides a kilogram per day emission rate as opposed to DPM concentrations and health risks. According to the CalEnviroScreen 4.0 report⁷, DPM emissions for on-road sources were generated **using CARB's on-road emissions model, EMFAC2017**, to calculate county-wide estimates of DPM emissions for a typical July weekday. Non-road sources were calculated using county-wide estimates of DPM for a July weekday, extracted **from CARB's emission inventory forecasting system**.

Due to the overly conservative nature of using a maximum daily DPM emission rate for a 70-year analysis, CalEnviroScreen data was not analyzed for the selected locations. An initial model was run using CalEnviroScreen emission rates for the previous iteration of this report, **analyzed using CARB's HARP** program, and the cancer risk values were more than an order of magnitude higher than the EPA NATA assessment. An assessment utilizing CalEnviroScreen data would have generated unrealistically high baseline cancer risks.

⁷ Available at: <https://oehha.ca.gov/media/downloads/calenviroscreen/report/calenviroscreen40reportf2021.pdf>

4. TOXICITY OF PETROLEUM DIESEL AND BIODIESEL EXHAUST

4.1 Toxicity of Petroleum-Derived Diesel Exhaust

4.1.1 U.S. EPA

As mentioned in Section 3 of this report, U.S. EPA does not have an explicit cancer risk value for total DPM. Instead, the cancer risk values of the individual components are utilized to generate an estimate of excess cancer cases. The individual components of DPM considered carcinogenic include the following compounds:

- Acetaldehyde
- Arsenic
- B[a]anthracene
- Benzene
- Beryllium
- 1,3-Butadiene
- Cadmium
- Cr(VI)
- Chrysene
- D[a,h]anthracene
- Formaldehyde
- In[1,2,3-cd]pyr
- Lead
- Naphthalene
- Nickel
- PAHs (as Benzo(a)pyrene)

The unit risk values of these pollutants are listed in Table 4-1.

4.1.2 CARB

Unlike EPA, OEHHA and CARB have generated a unit risk value for DPM exhaust. It should be noted, these unit risk values cannot be compared directly as the DPM unit risk is compared against 100% of diesel emissions, whereas there are varying degrees of composition for other compounds in diesel exhaust.

Table 4-1. Comparison of U.S. EPA and CARB Diesel Exhaust Toxicity Values

Compound	U.S. EPA Unit Risk ($\mu\text{g}/\text{m}^3$) ⁻¹	CARB Unit Risk ($\mu\text{g}/\text{m}^3$) ⁻¹
Diesel Exhaust	N/A	0.0003
Acetaldehyde	0.0000022	0.0000027
Arsenic	0.0043	0.0033
B[a]anthracene	0.00006	0.00011
Benzene	0.0000078	0.000029
Beryllium	0.0024	0.0024
1,3-Butadiene	0.00003	0.00017

Compound	U.S. EPA Unit Risk ($\mu\text{g}/\text{m}^3$) ⁻¹	CARB Unit Risk ($\mu\text{g}/\text{m}^3$) ⁻¹
Cadmium	0.0018	0.0042
Chrysene	0.0000006	0.000011
Cr(VI)	0.012	0.15
D[a,h]anthracene	0.0006	0.0012
Formaldehyde	0.000013	0.000006
In[1,2,3-cd]pyr	0.00006	0.00011
Lead	0	0.000012
Naphthalene	0.000034	0.000034
Nickel	0.00024	0.00026
PAHs (as Benzo(a)pyrene)	0.0006	0.0011

4.2 Toxicity of Biodiesel Exhaust

The combustion of biodiesel in compression ignition (diesel) engines is generally understood to produce significantly lower emissions of carcinogenic diesel particulate matter (DPM) compared to conventional petroleum-based ultra-low sulfur diesel (ULSD) in older engines that are not equipped with diesel particulate filters (DPFs) or diesel oxidation catalysts (DOCs). Trinity conducted a literature review of relevant studies focusing on the reductions of DPM emissions exhibited by various categories and ratings of diesel engines for both on-road and nonroad vehicles and equipment. The literature review confirmed that reductions in DPM emissions are observed in older-technology diesel engines. These are effectively engines certified to 2006 and earlier on-highway heavy-duty compression ignition standards and Tier 3 and earlier nonroad compression ignition (NRCI) engine standards. These engines make substantial contributions to total DPM emissions from on- and non-road sources as well as public exposure to DPM which is the focus of this study.

The sponsor of this study, the Clean Fuels Alliance America (CFAA), formerly known as the National Biodiesel Board, directed Trinity to assume that the reduction in DPM emissions due to the use of B100 in **older Diesel engines is 72% based on emissions data for a specific engine contained in CARB's Biodiesel Characterization and NOx Mitigation Study (October 2011).**⁸ Trinity, as directed by CFAA, applied this reduction percentage to older-technology on-road and nonroad engines as described in this report to produce an upper-bound (i.e., maximum) estimate of the potential benefits that biodiesel can provide in the communities that are the focus of this study. It is again important to note that no reductions due to biodiesel use were assumed to occur with newer technology diesel engines equipped with factory DOCs or DPFs.

⁸ See (CARB, 2011), Table ES-9 for PM reductions exhibited by a 2000 model year Caterpillar C-15 engine operating on soy-based and animal-based B100.

5. HEALTH RISK ASSESSMENT METHODOLOGY

The following subsections describe how health risk values were determined for each type of health risk assessment.

5.1 NATA Health Risk Assessment Methodology

The NATA provides overall DPM concentrations, individual source contributions to the total DPM concentration, and individual source risk values. Because DPM risks are not calculated directly, the baseline DPM risks for this evaluation are assumed to equal only those risks from sources that explicitly emit DPM. Therefore, NATA derived DPM risks represent a low estimate of DPM cancer risks.

5.2 NATA/HARP Hybrid Risk Assessment Methodology

Because NATA provides DPM concentrations on a census tract basis, those values are able to be evaluated **in CARB's HARP program to determine cancer risks with OEHHA-specific unit risk values**. The overall cancer risk from DPM is approximately 100 times **higher using OEHHA's unit risk value as opposed to the** cumulative risk from the individual component unit risk values derived by EPA.

For all HARP runs, a population-wide assessment was conducted using a 70-year exposure period, with all default values as assigned by the OEHHA Risk Assessment Guidelines⁹.

5.3 Site-Specific Health Risk Assessments

Section 6 outlines how each site-specific health risk assessment was modeled. For all HARP runs, a population-wide assessment was conducted using a 70-year exposure period, with all default values as assigned by the OEHHA Risk Assessment Guidelines¹⁰.

5.4 Valuation of Health Risk Benefits

The monetary valuation of health benefits from using biodiesel was evaluated using USEPA's **Environmental Benefits Mapping and Analysis Program - Community Edition (BenMAP)**, Version 1.5.8.¹¹ BenMAP is capable of calculating the reduction in incidence or prevalence of negative health impacts associated with a corresponding reduction in ambient PM_{2.5} concentration. BenMAP also allows for the valuation of these reductions based on the use of user-specified valuation functions.

The methodology contained within BenMAP is routinely used by CARB to estimate the health benefits of various rulemaking activities aimed at reducing PM_{2.5} emissions.¹² For this reason, the assumptions and **model inputs that were selected for this analysis are based on CARB's methodology as described in detail in Appendix J of the California Truck and Bus Initial Statement of Reasons**¹³ (except as noted).

⁹ <https://oehha.ca.gov/media/downloads/cnr/2015guidancemanual.pdf>

¹⁰ Ibid.

¹¹ <https://www.epa.gov/benmap>

¹² <https://ww2.arb.ca.gov/resources/documents/carbs-methodology-estimating-health-effects-air-pollution>

¹³ <https://ww3.arb.ca.gov/regact/2010/truckbus10/correctedappj.pdf>

5.4.1 Geography, Incidence/Prevalence, and Population

For each community, benefits were calculated on a census tract basis, with total benefits equaling the aggregation of all census tracts within each analysis community. Incidence/prevalence rates were selected from BenMAP default data sets. The population dataset was derived from U.S. Census data at the smallest geographic unit, which is the county level. The analysis selected included a population growth estimate to reflect the 2020 calendar year.

5.4.2 Health Impacts

The health impacts analyzed consist of the following:

- > Acute Myocardial Infarctions (AMIs, or heart attacks)
- > Asthma Symptoms – Albuterol Use
- > Emergency Room (ER) Visits – All Cardiac Outcomes
- > ER Visits – Respiratory
- > Hospital Admissions (HA) – All Respiratory
- > HA – **Alzheimer’s Disease**
- > HA – Cardio-, Cerebro-, and Peripheral Vascular Disease
- > HA – **Parkinson’s Disease**
- > HA – Respiratory-2
- > Incidence – Asthma
- > Incidence – Hay Fever/Rhinitis
- > Incidence – Lung Cancer
- > Incidence – Out of Hospital Cardiac Arrest
- > Incidence – Stroke
- > Minor Restricted Activity Days
- > Mortality – All Causes
- > Premature Mortality (all causes)
- > **Acute respiratory symptoms resulting in “minor restricted activity days”**
- > Work loss days

The above health impacts, or “endpoints” are those routinely used by CARB during their rulemaking, and hence were used for this analysis.

For each endpoint, BenMAP requires the user to select one or more health impact functions. Each health impact function option represents a technical study reflecting the relationship between PM_{2.5} concentrations and the health impact “endpoint” that is being studied. **With regard to the above health impact endpoints, the studies relied upon were selected based on those used in the CARB analyses previously stated, to the degree possible.**

Specifically, for the endpoint of “premature death” (which includes cancer deaths), the analysis relied upon the study *Pope et al., 2002*, which is also the study CARB has primarily relied upon. For the asthma exacerbation endpoint, the study CARB relied upon is not included within BenMAP. Hence, all available studies related to asthma endpoints were “pooled” using that functionality of BenMAP. For acute respiratory symptoms resulting in minor restricted activity days, the analysis relied upon the study *Ostro and Rothschild, 1989*. And finally, for work loss days, the study *Ostro, 1987* was selected, which is also a study CARB has primarily relied upon.

It is important to note that the endpoint of premature deaths calculated by BenMAP is not equivalent to the cancer burden values discussed in this report. This is because the endpoint of premature death encompasses all causes, including both lung cancer and ischemic heart disease. In contrast, the metric of cancer burden includes all types of cancers attributed to PM_{2.5} exposure. Likewise, cancer burden relates to incidence rate of cancer, which is not the same as the premature death endpoint. Many cancer cases do not result in death, and hence, cancer burden reductions will always be higher than avoided premature deaths calculated by BenMAP.

5.4.3 Valuation Functions

Valuation functions assign a value to each health impact “endpoint.” Of the above health impacts, reduced premature mortality will always dominate the overall benefit value under any scenario. However, to document the use of BenMAP, it is important to document the valuation functions used for each endpoint included in this project.

- > For the endpoint of premature mortality, the BenMAP **standard valuation function “based on 26 value-of-life studies” was selected.**
- > For the health impact endpoint of asthma exacerbation, all of the available health impact functions within BenMAP were pooled to derive a result.
- > For the acute respiratory endpoint of minor restricted activity days, the standard EPA valuation **function of “WTP: 1 day, CV studies” was selected.**
- > For the endpoint of work loss days, the standard EPA valuation function of median work loss days, county specific was selected.

5.4.4 General Valuation Results

Specific results are provided below in Section 6, for each community. In a general, it is noted that the overall value of benefits is sensitive to (1) the extent of geographic area analyzed, and (2) the population living within that same geographic area. That is, analyses performed over a broader area, and encompassing a greater population, will produce greater benefits.

6. HEALTH RISK ASSESSMENT RESULTS

6.1 District of Columbia

6.1.1 NATA Health Risks

The subsections below review the NATA data available for the DC community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-1 shows the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

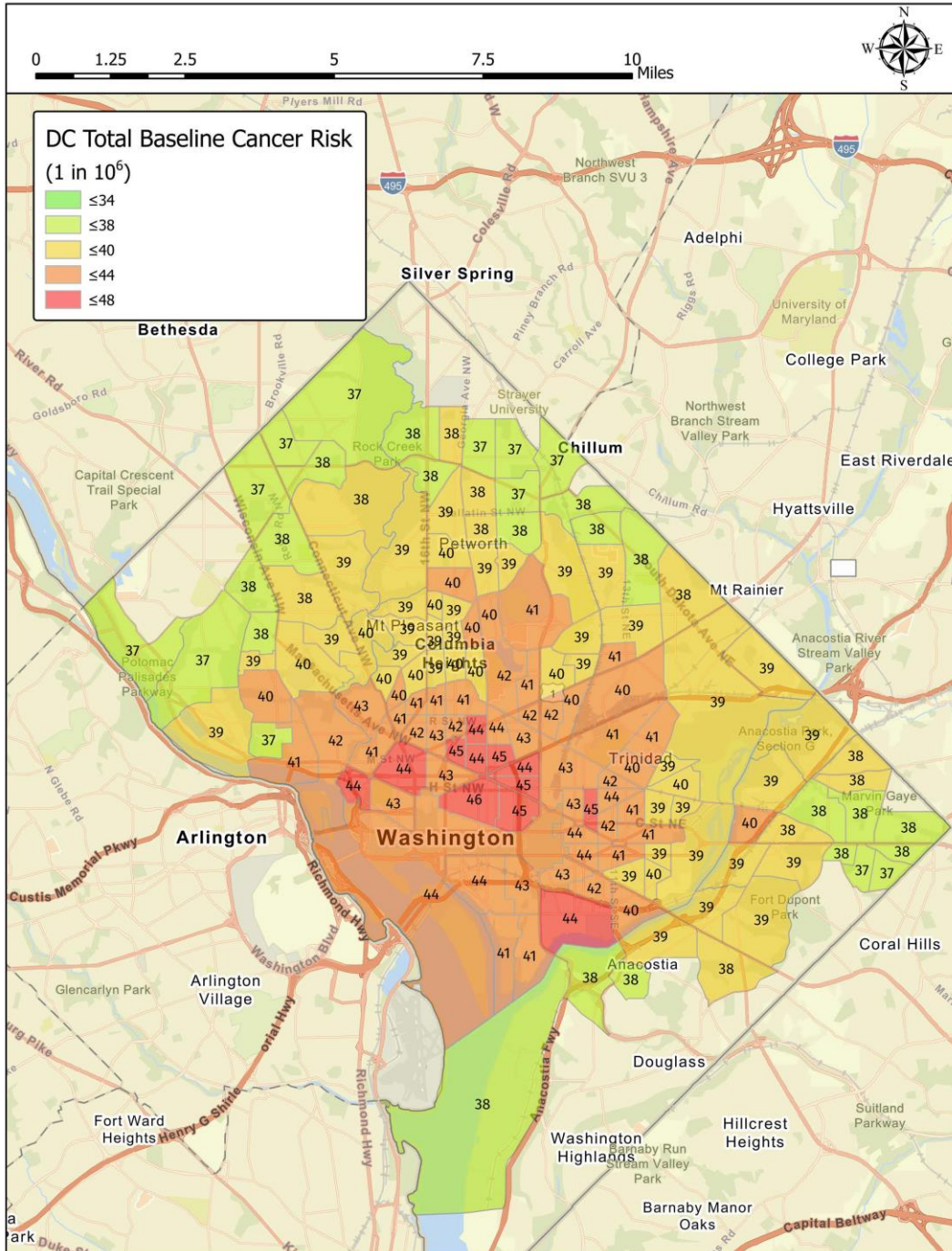
Figure 6-2 shows those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-3 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the DC area.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the DC area.

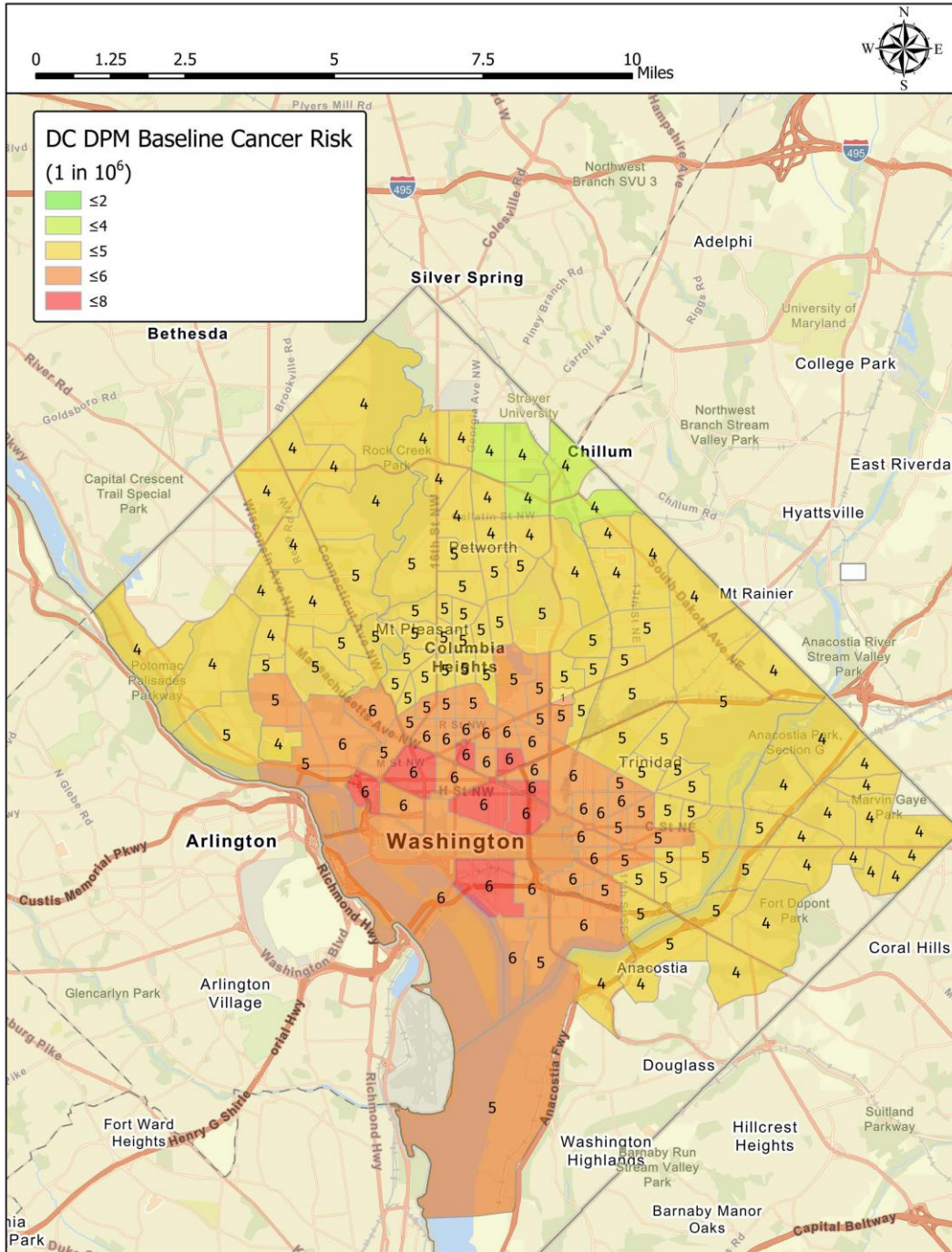
6.1.1.1 NATA Risk Data

Figure 6-1. DC Baseline NATA Total Cancer Risks



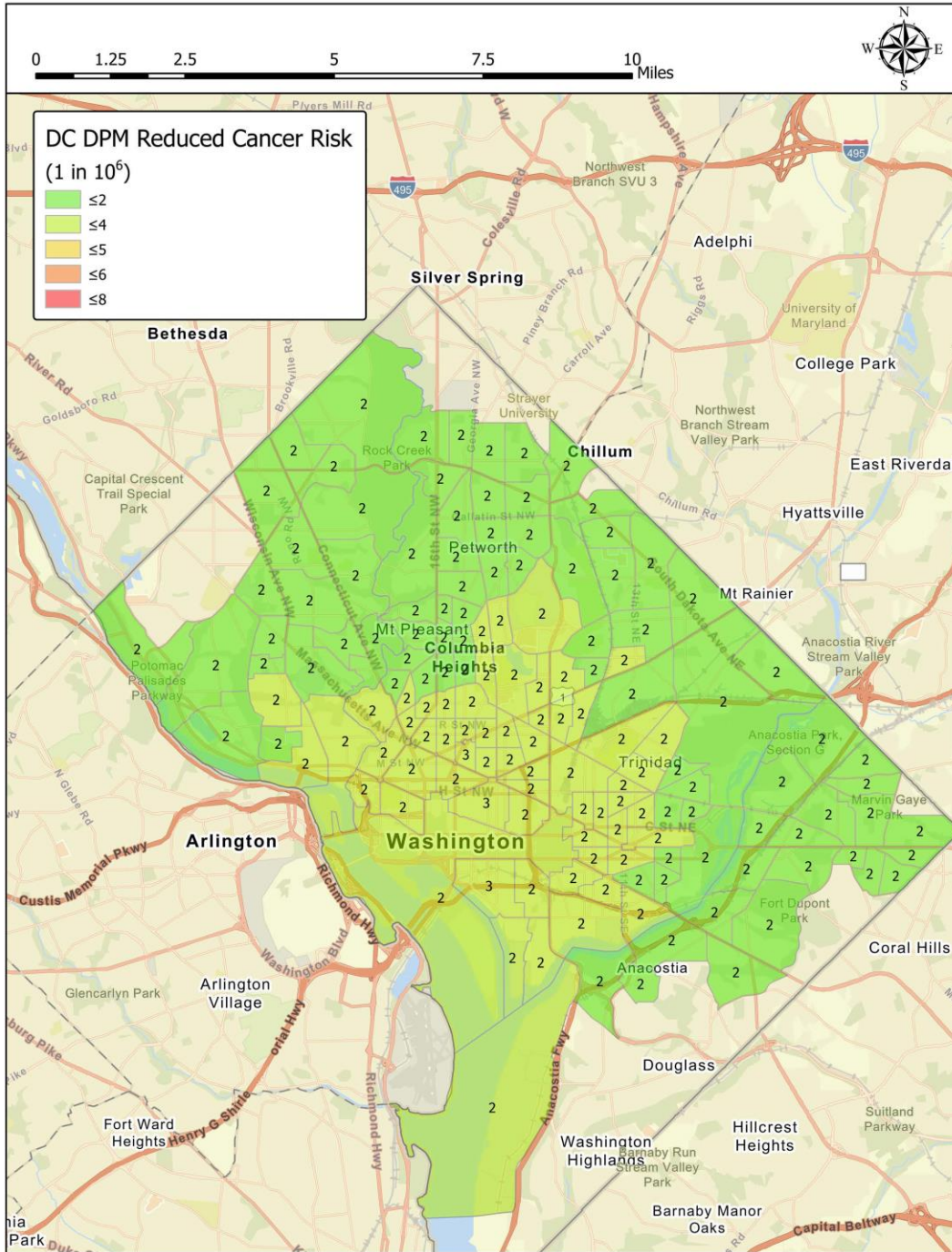
According to the NATA, the maximum baseline cancer risk in the DC area is 45.6 cancer cases per million residents for census tract 11001005800, with a population of 2,830 residents. When accounting for all of the communities assessed, the total cancer burden for the DC area is 20 cancer cases expected over a 70-year timeline among a total community population of 498,507.

Figure 6-2. DC Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the DC area is 6 cancer cases per million residents for census tract 11001005800, with a population of 2,830 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the DC area is 2 cancer cases expected over a 70-year timeline among a total community population of 498,507.

Figure 6-3. DC Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the DC area becomes 3 cancer cases per million residents for census tract 11001005800, with a population of 2,830 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the DC area becomes 1 cancer case expected over a 70-year timeline among a total community population of 498,507.

6.1.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB's HARP program with OEHHA cancer unit risk values. The data is outlined in the following order:**

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program.**

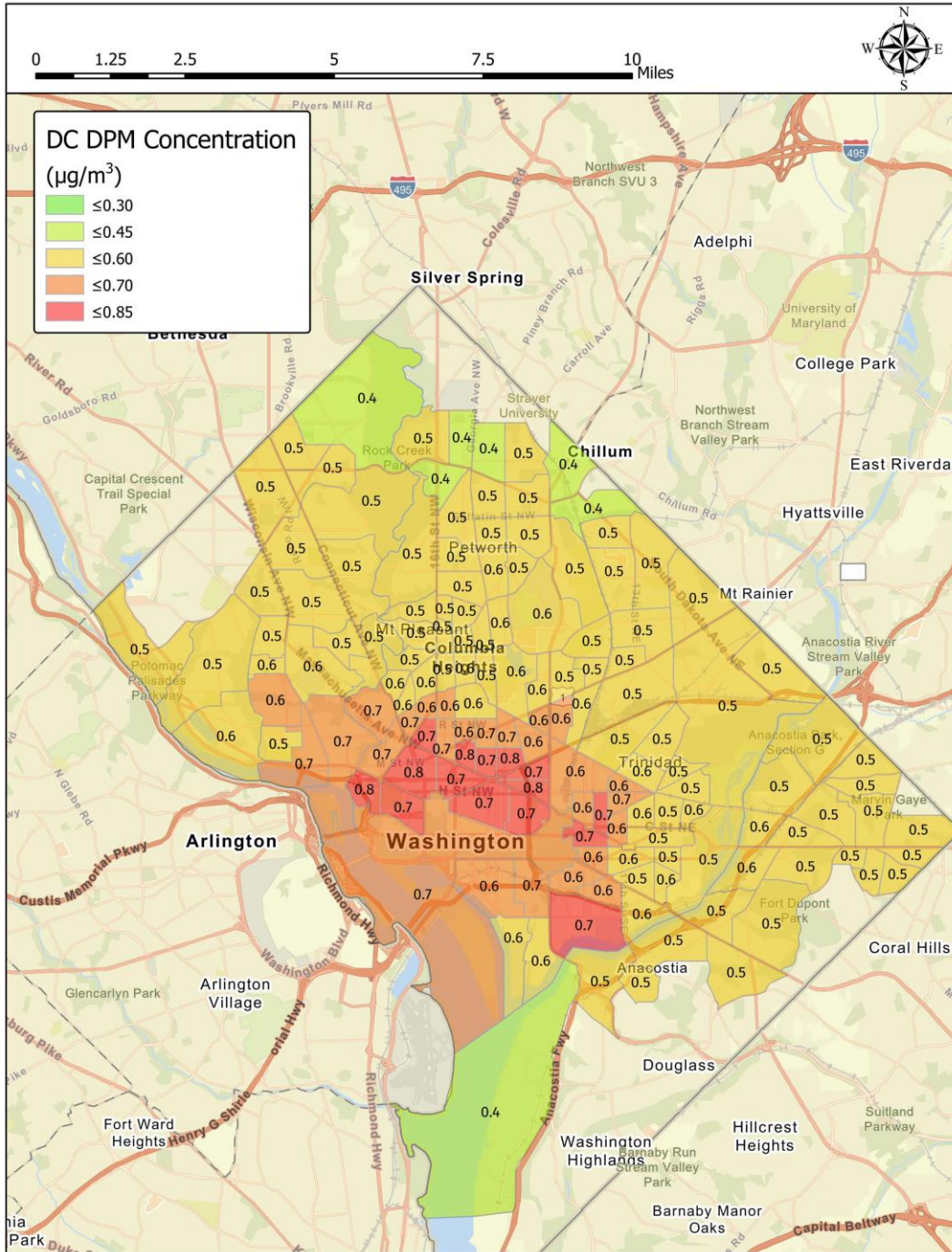
Figure 6-4 shows the baseline DPM concentrations provided by the NATA.

Figure 6-5 shows the baseline DPM-specific cancer risks as determined using the NATA concentration values **and CARB's HARP program.**

Figure 6-6 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the DC area.

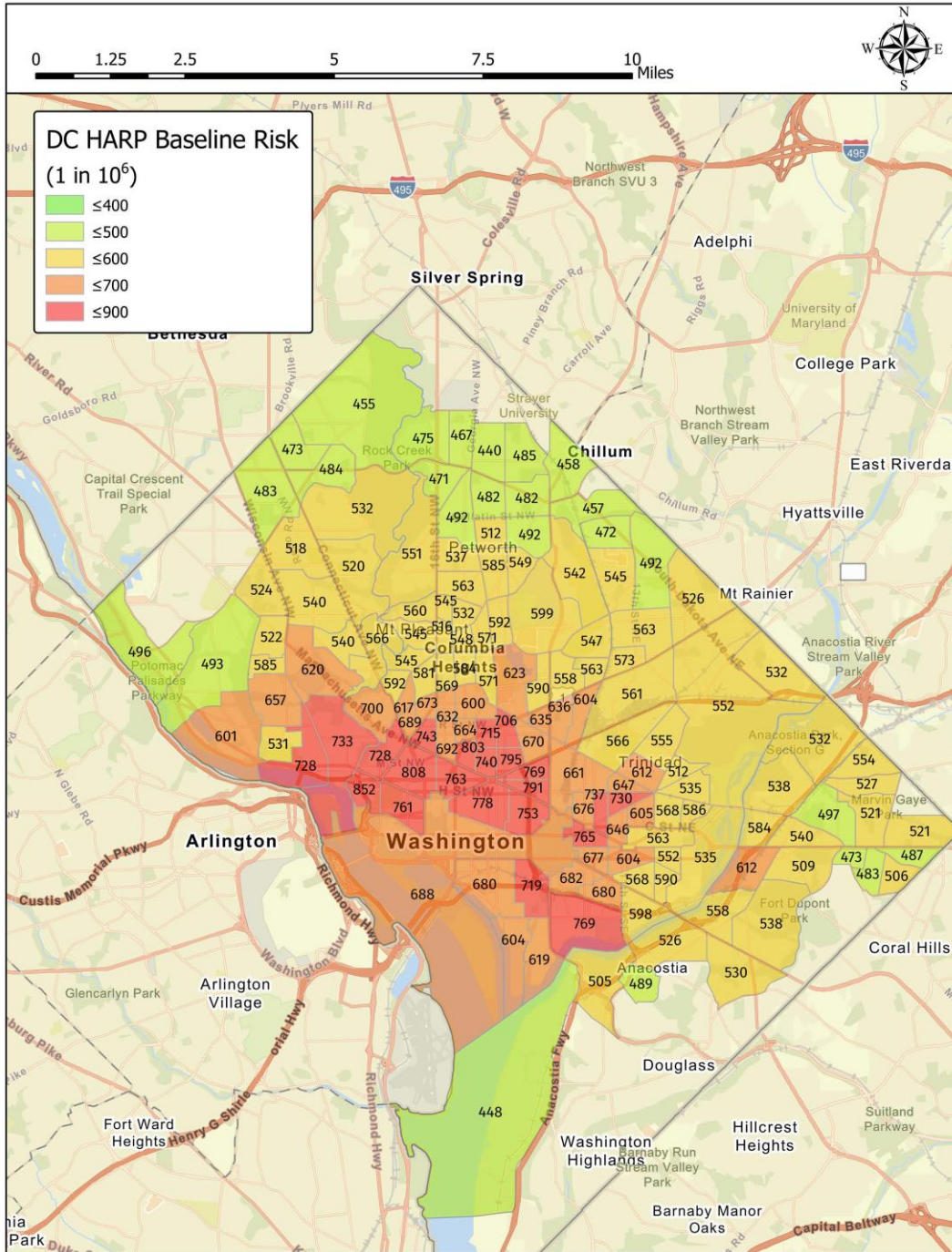
Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the DC area.

Figure 6-4. DC Baseline NATA DPM Concentrations



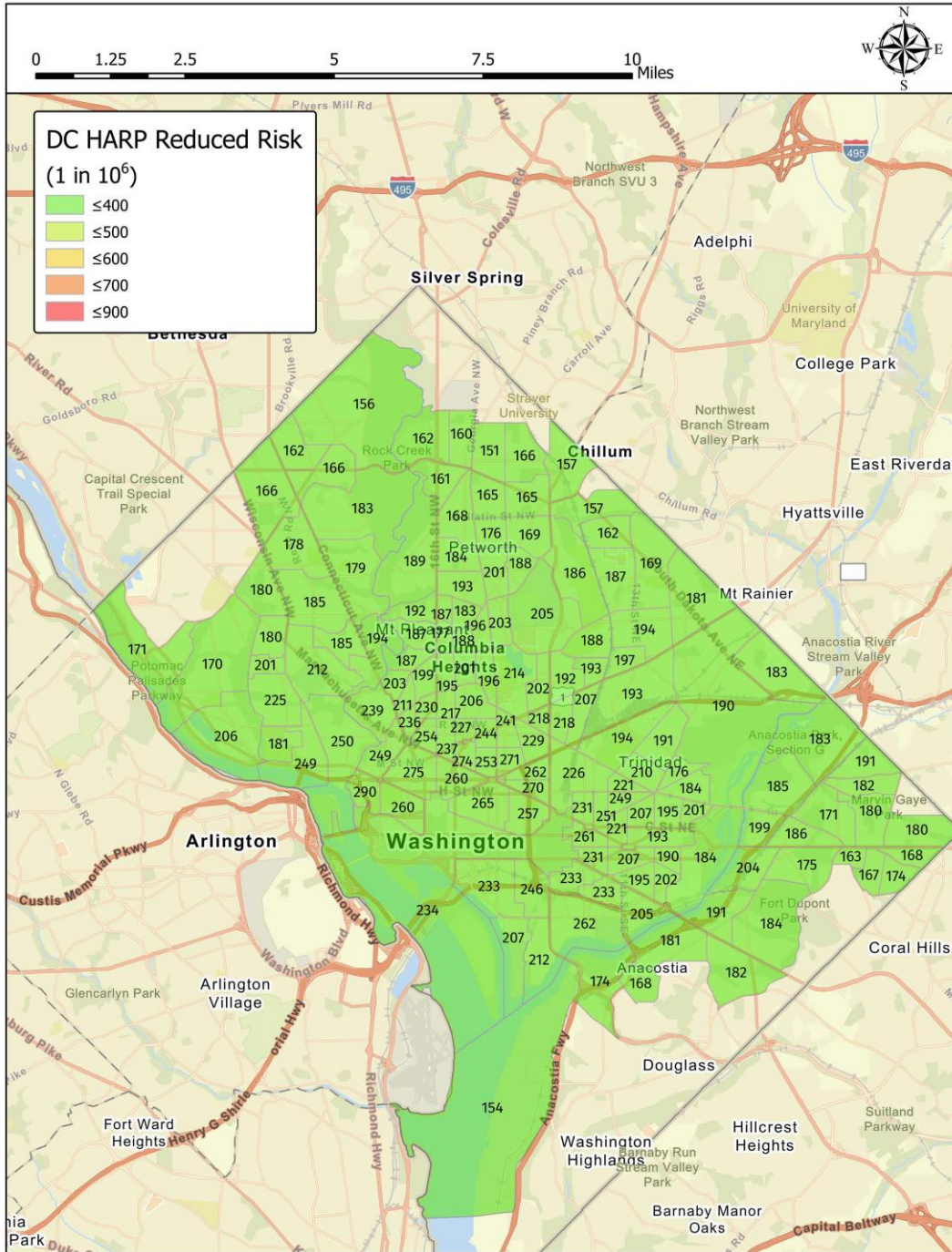
According to the NATA, the maximum baseline DPM concentration in the DC area is 0.81 $\mu\text{g}/\text{m}^3$ for census tract 11001005600, with a population of 6,756 residents. The average DPM concentration of the DC area is 0.56 $\mu\text{g}/\text{m}^3$.

Figure 6-5. DC Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the DC area is 852 cancer cases per million residents for census tract 11001005600, with a population of 6,756 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the DC area is 293 cancer cases expected over a 70-year timeline among a total community population of 498,507.

Figure 6-6. DC Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the DC area becomes 290 cancer cases per million residents for census tract 11001005600, with a population of 6,756 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the DC area becomes 100 cancer cases expected over a 70-year timeline among a total community population of 498,507.

6.1.2 DC Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for DC. The following sources were utilized to generate the HRA.

- District of Columbia Department of Transportation (DDOT) – Traffic Counts (2019 Average Annual Daily Traffic)
- Virginia Department of Transportation – Traffic Counts (2019 Average Annual Daily Traffic)

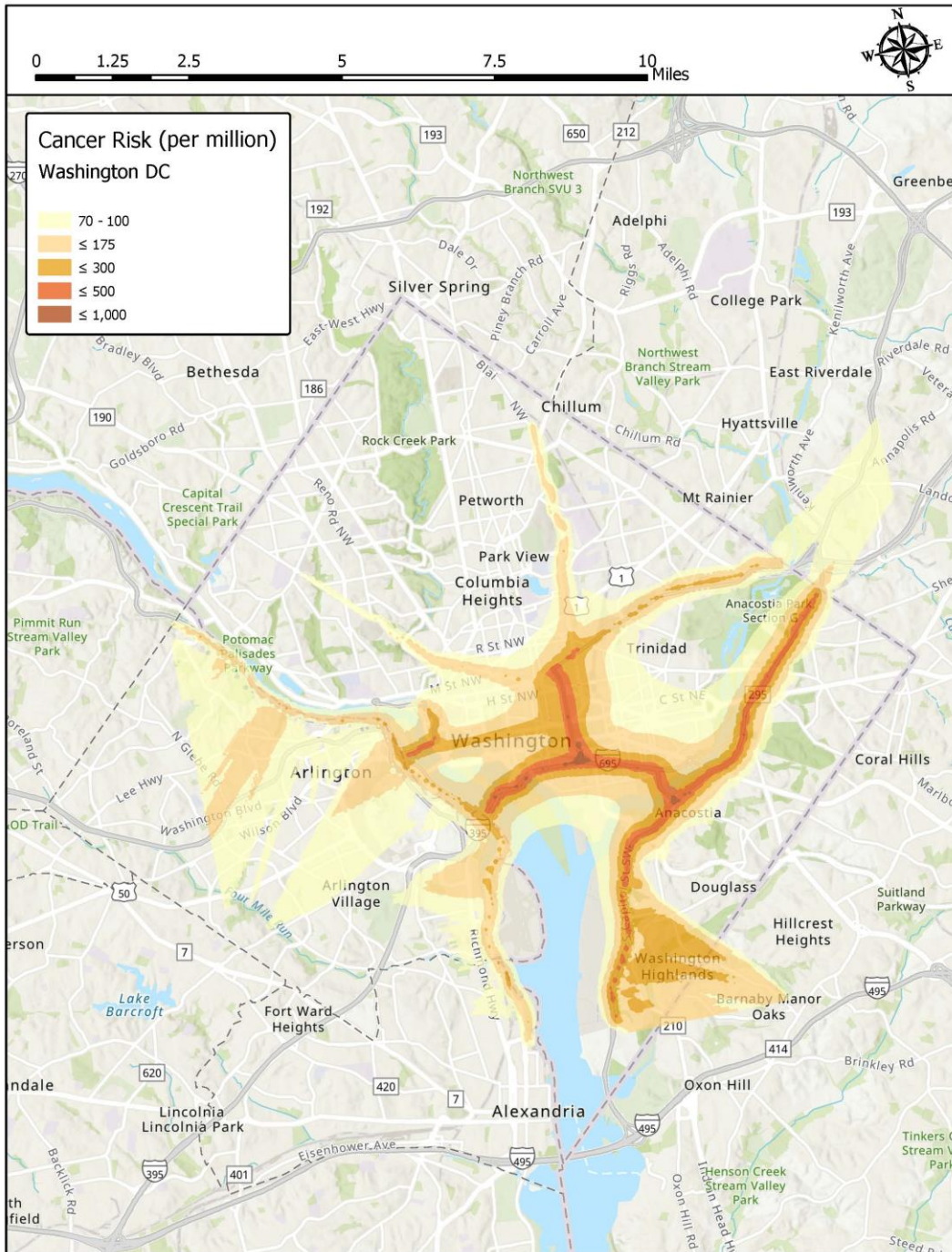
The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-1.

Table 6-1. DC Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
I-295	I-295 – 117,243 AADT	9,694	59.7%
I-695	I-695 – 133,839 AADT	2,604	59.7%
I-395	I-395 – 150,754 AADT	5,210	59.7%
US 50	US 50 – 48,473 AADT	3,677	59.7%
I-66	I-66 – 60,389 AADT	723	59.7%
Mass Ave	Massachusetts Avenue – 30,101 AADT	1,278	59.7%
N Capitol	North Capitol Street – 39,676 AADT	1,885	59.7%
GW Pkwy	George Washington Parkway – 44,857 AADT	4,624	59.7%

These sources were modeled with unit emission rates in AERMOD, and the Table 6-1 listed emission rates were input into CARB's HARP software to determine cancer risks from the DPM concentrations determined by AERMOD. While dispersion characteristics remained the same between baseline and reduced modeling scenarios, emission rates were reduced according to the number of "old technology" engines combusting diesel, based on source type. The table above shows the Proportion of "Old Technology" Engine Emissions where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

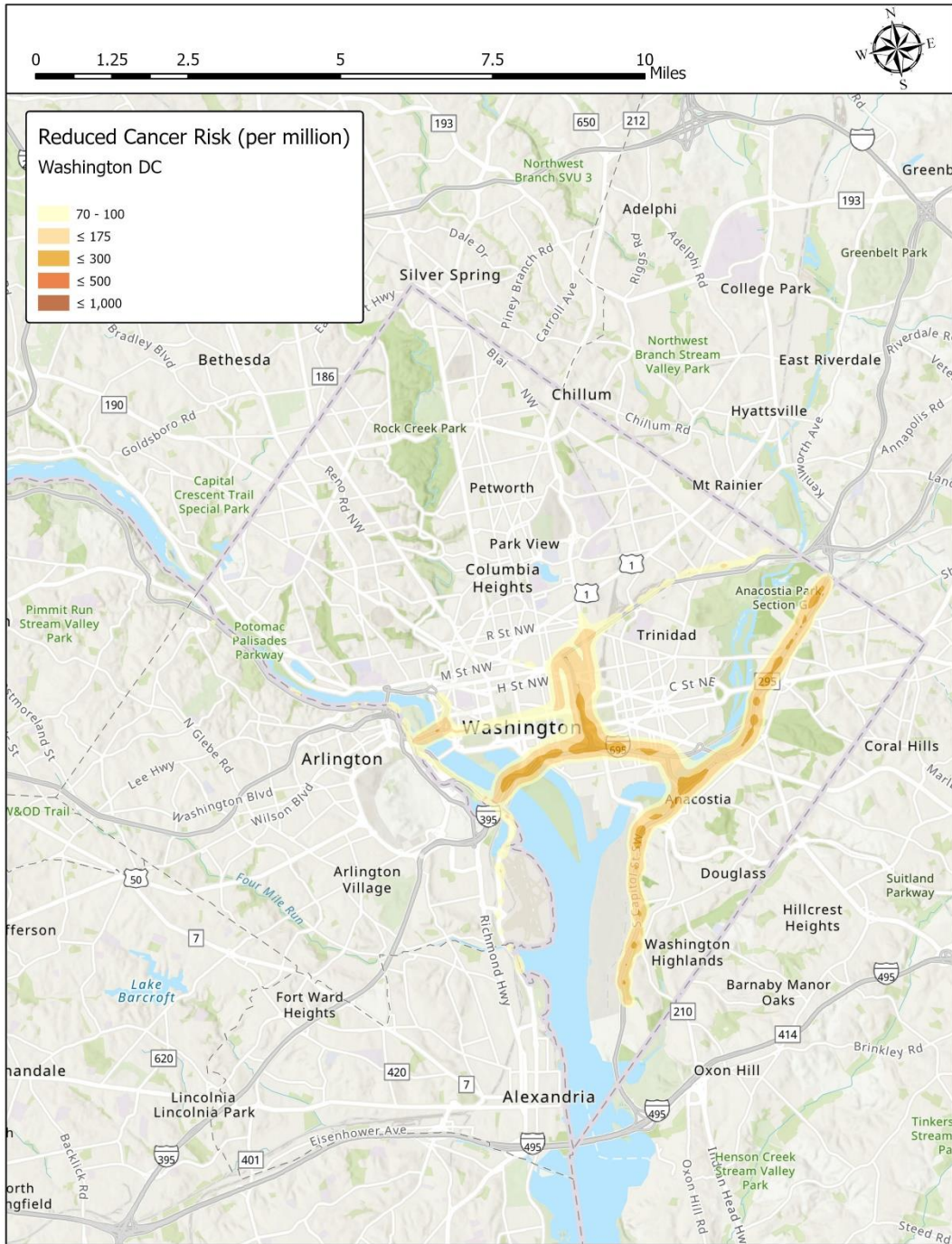
Figure 6-7. DC Baseline Health Risk Assessment Isopleths



The site-specific HRA shows that the point of maximum impact (PMI) is slightly lower than the NATA/HARP evaluation, with an impact of 793 cancer cases per million residents. This PMI does not occur at a residential receptor, and does not represent an actual risk to residences in the area. The MEIR occurs at 330,112 m E, and 4,306,424 m N (NAD 83, UTM Zone 18), with a baseline risk of 640 cancer cases per million residents. This MEIR is higher than the NATA/HARP hybrid risks evaluated for that census tract

(11001009604) with a risk of 584 in a million, demonstrating the inability of the NATA to determine local maxima in cancer risks.

Figure 6-8. DC Reduced Health Risk Assessment Risk Isopleths



The reduced cancer risk PMI and MEIR are 320 and 258 in one million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 382 in one million at the MEIR.

6.1.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-2 below.

Table 6-2. DC Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	100.6	\$3,303,129
Asthma Symptoms - Albuterol use	12,987	\$4,488
ER visits – All Cardiac Outcomes	12.3	\$14,299
ER visits – Respiratory	28.1	\$24,598
HA – All Respiratory	3.1	\$54,807
HA - Alzheimer's Disease	10.5	\$133,287
HA - Cardio- Cerebro- and Peripheral Vascular Disease	4.3	\$68,492
HA - Parkinson's Disease	1.7	\$22,980
HA - Respiratory-2	0.6	\$0
HA - Respiratory-2 HA – All Respiratory	3.7	\$0
Incidence - Asthma	98.8	\$4,414,345
Incidence - Hay Fever/Rhinitis	619.3	\$371,503
Incidence - Lung Cancer	4.7	\$59,160
Incidence - Out of Hospital Cardiac Arrest	0.6	\$20,552
Incidence - Stroke	1.9	\$63,297
Minor Restricted Activity Days	33,036	\$2,298,710
Mortality - All Cause	32.1	\$249,689,228
Work Loss Days	5,680	\$1,467,432
Total		\$262,010,306

6.2 Phoenix, Arizona

6.2.1 NATA Risks

The subsections below review the NATA data available for the Phoenix area. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-9 shows the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

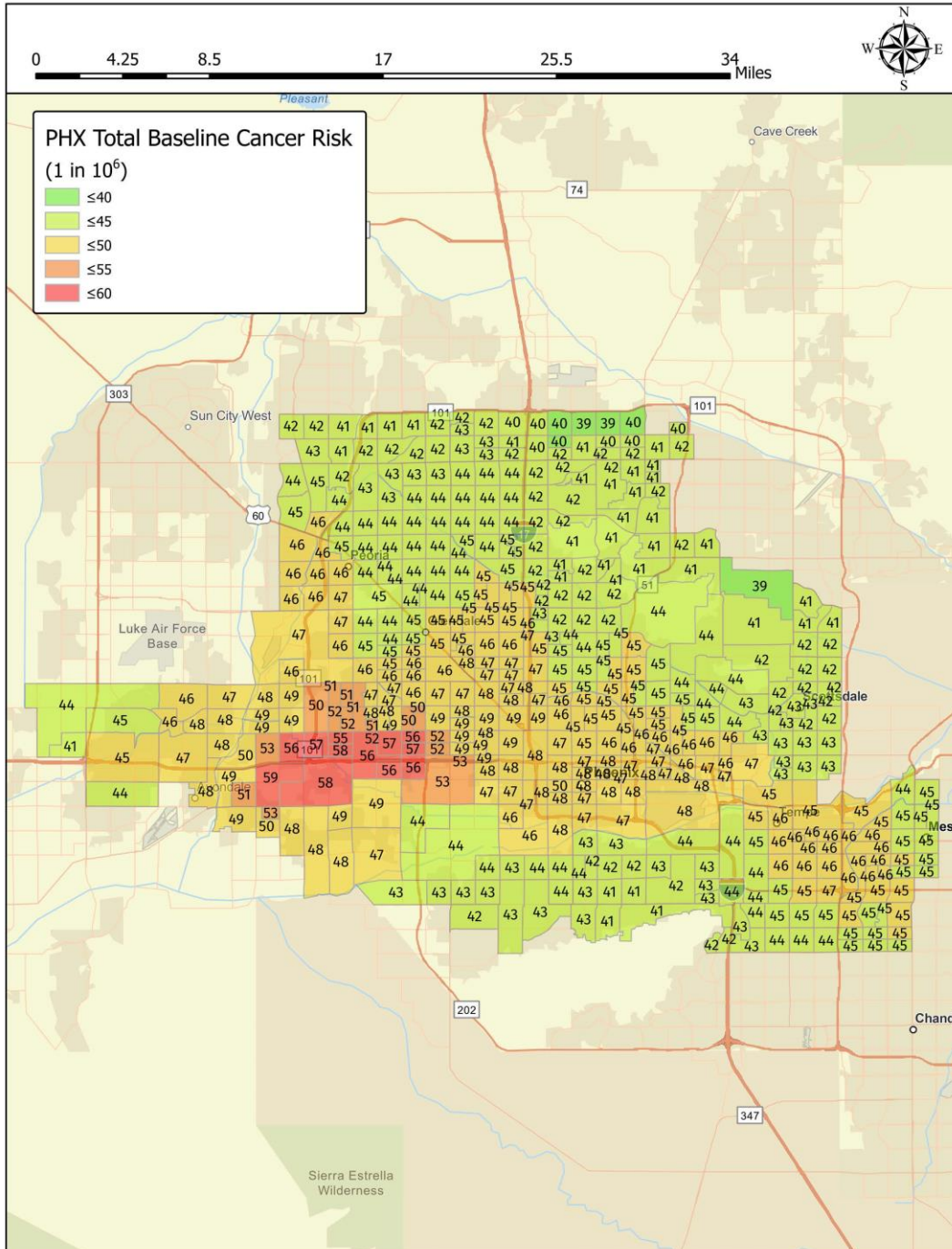
Figure 6-10 shows those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-11 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Phoenix community.

Because the NATA analysis utilized EPA specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the Phoenix community.

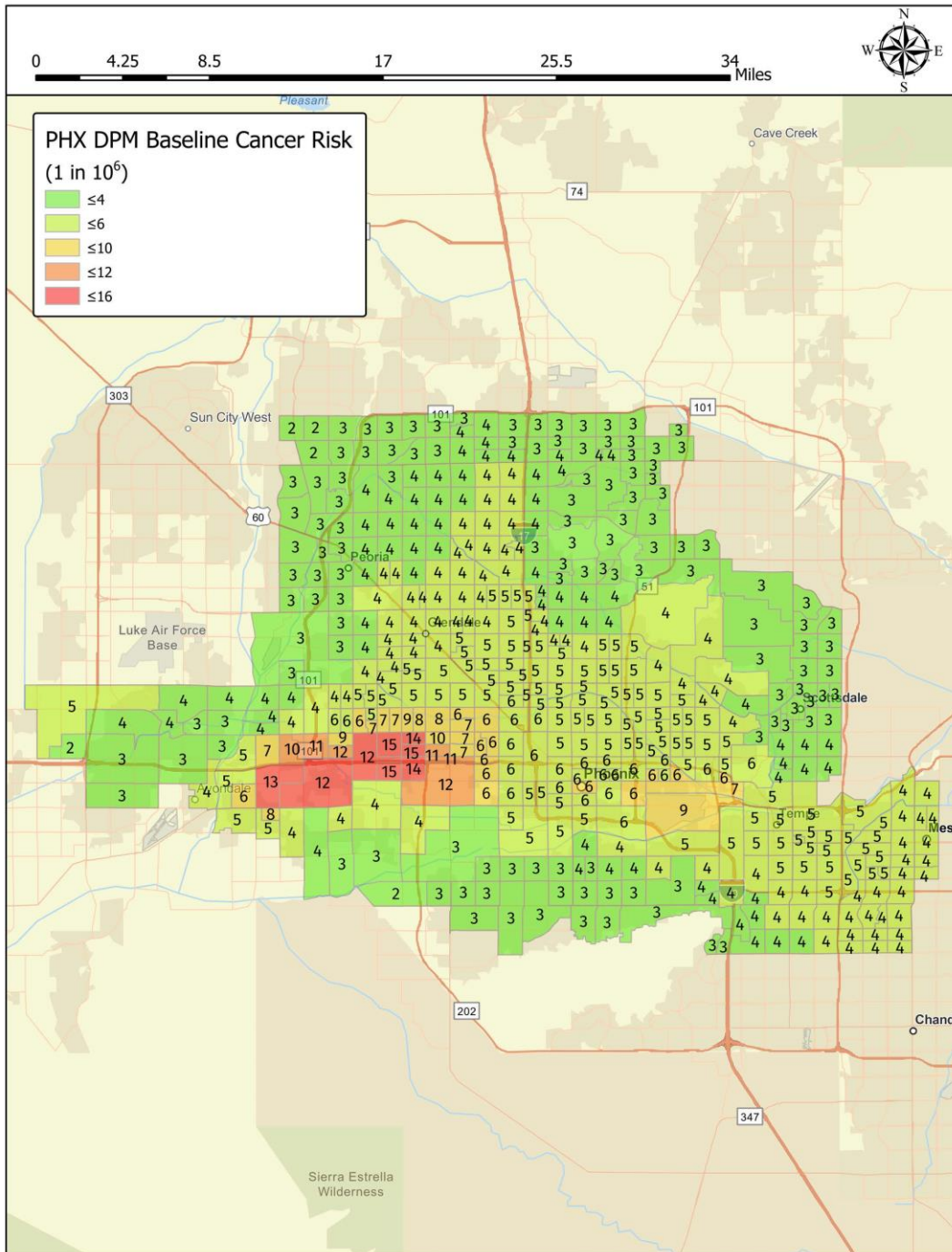
6.2.1.1 NATA Risk Data

Figure 6-9. Phoenix Baseline NATA Total Cancer Risks



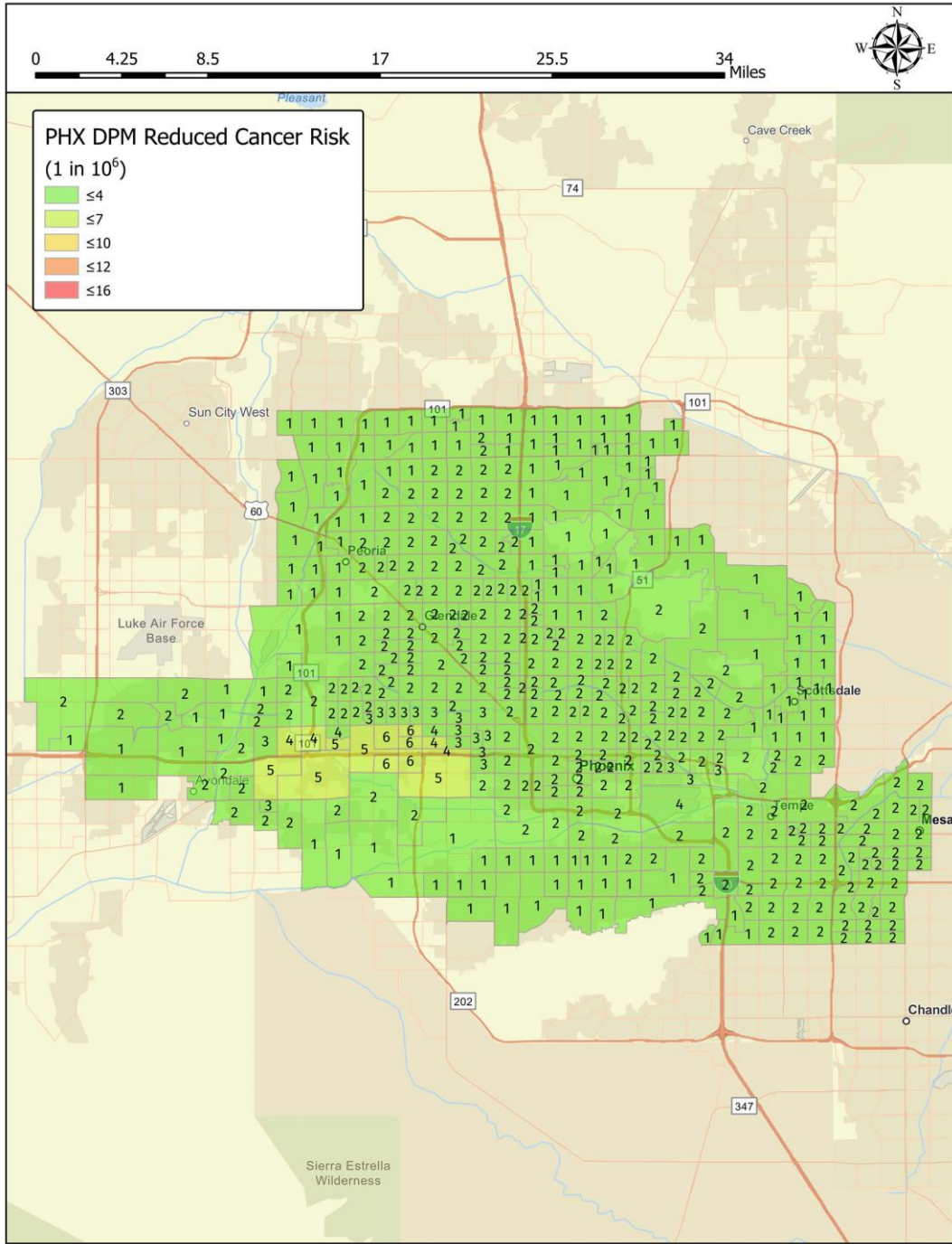
According to the NATA, the maximum baseline cancer risk in the Phoenix community is 59.32 cancer cases per million residents for census tract 04013082027, with a population of 3,978 residents. When accounting for all of the communities assessed, the total cancer burden for the Phoenix community is 89 cancer cases expected over a 70-year timeline among a total community population of 1,963,841.

Figure 6-10. Phoenix Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the Phoenix community is 15 cancer cases per million residents for census tract 04013112502, with a population of 5,397 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Phoenix community is 9 cancer case expected over a 70-year timeline among a total community population of 1,963,841.

Figure 6-11. Phoenix Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Phoenix community becomes 6 cancer cases per million residents for census tract 04013112502, with a population of 5,397 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Phoenix community becomes 4 cancer case expected over a 70-year timeline among a total community population of 1,963,841.

6.2.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB's HARP program with OEHHA cancer unit risk values. The data is outlined in the following order:**

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program.**

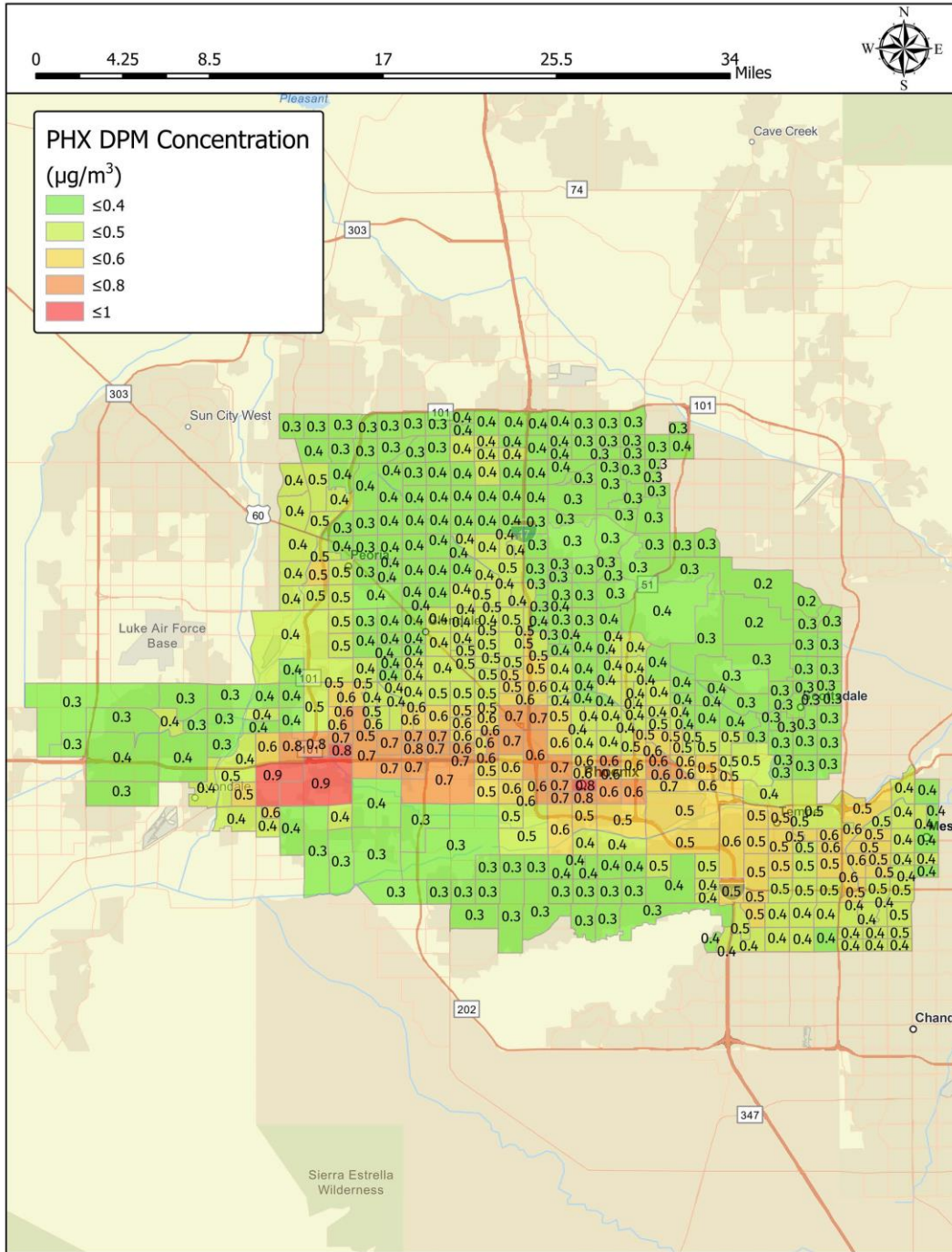
Figure 6-12 shows the baseline DPM concentrations provided by the NATA.

Figure 6-13 shows the baseline DPM-specific cancer risks as determined using the NATA concentration **values and CARB's HARP program.**

Figure 6-14 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Phoenix community.

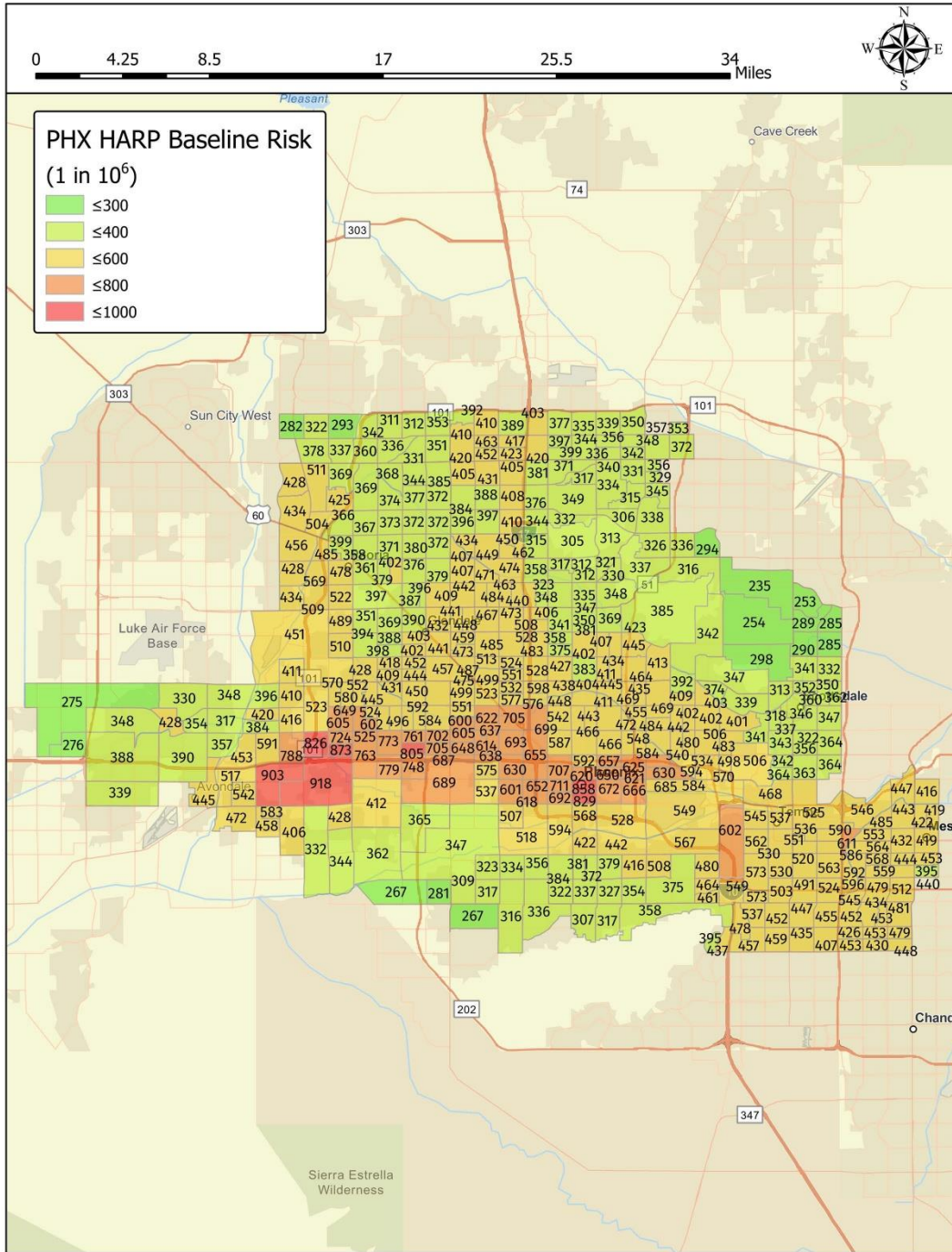
Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the Phoenix community.

Figure 6-12. Phoenix Baseline NATA DPM Concentrations



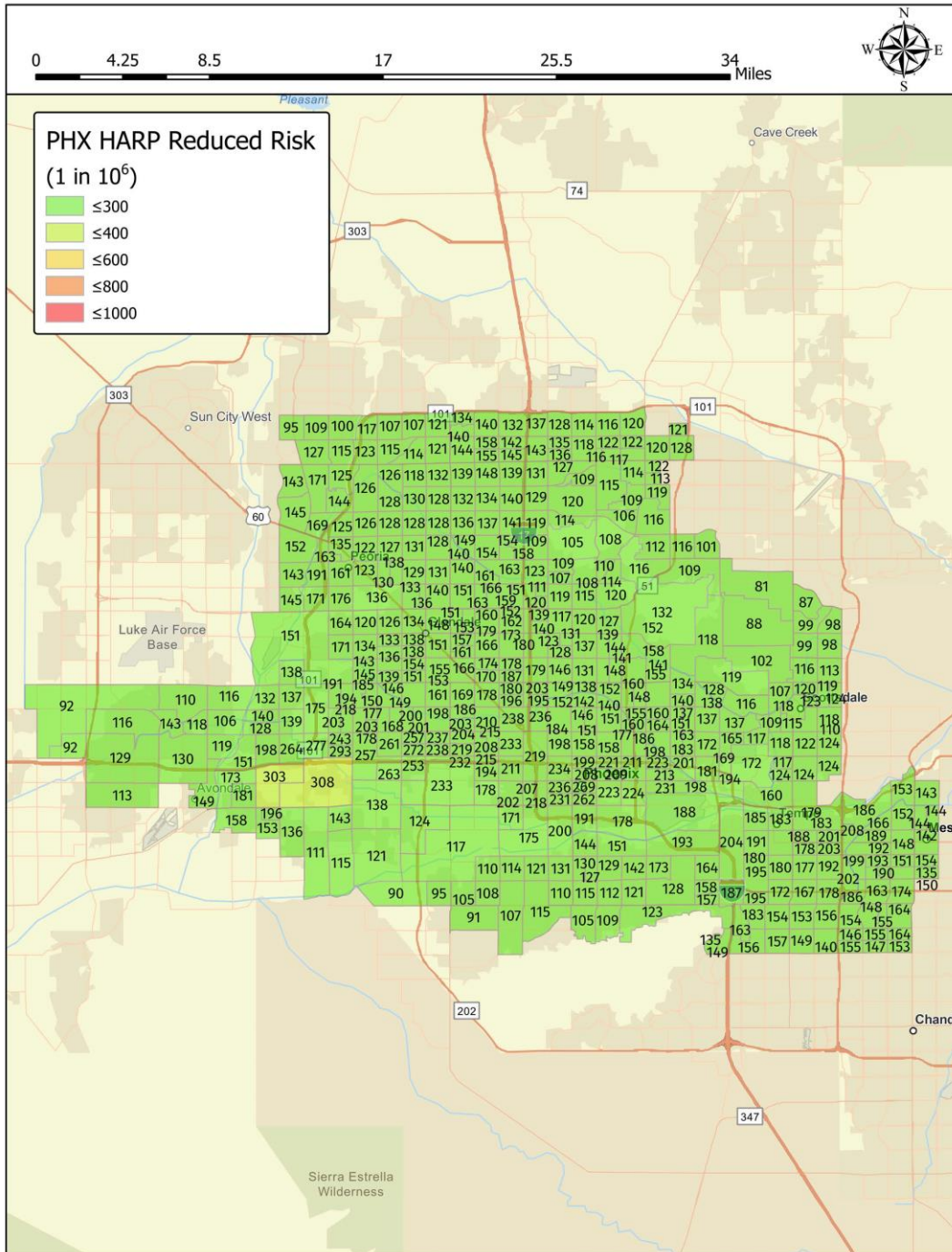
According to the NATA, the maximum baseline DPM concentration in the Phoenix community is $0.87 \mu\text{g}/\text{m}^3$ for census tract 04013083000, with a population of 6,545 residents. The average DPM concentration of the Phoenix community is $0.43 \mu\text{g}/\text{m}^3$.

Figure 6-13. Phoenix Baseline NATA/HARP Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the Phoenix community is 918 cancer cases per million residents for census tract 04013083000, with a population of 6,545 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Phoenix community is 887 cancer cases expected over a 70-year timeline among a total community population of 1,963,841.

Figure 6-14. Phoenix Reduced NATA/HARP Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Phoenix community becomes 308 cancer cases per million residents for census tract 04013083000, with a population of 6,545 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Phoenix community becomes 301 cancer cases expected over a 70-year timeline among a total community population of 1,963,841.

6.2.2 Phoenix Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Portland. The following sources were utilized to generate the HRA.

- Arizona Department of Transportation (ADOT) – Traffic Counts (2019 Average Annual Daily Traffic)

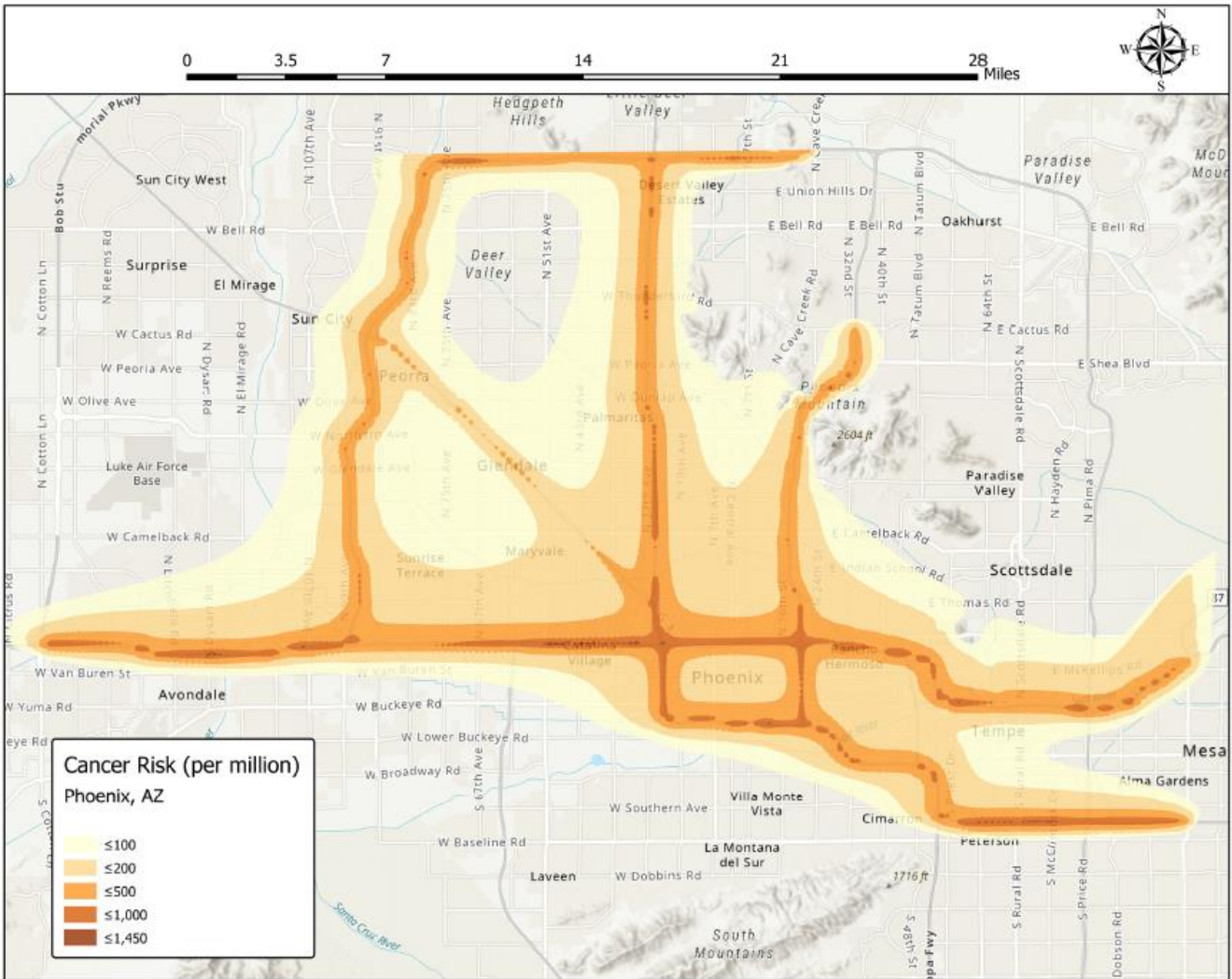
The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-3.

Table 6-3. Phoenix Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
202	State Route 202 – 179,724 AADT	63,353	59.7%
101	State Route 101 – 123,589 AADT	32,518	59.7%
60	U.S. 60 – 37,477 AADT	4,397	59.7%
17	I-17 – 180,689 AADT	60,713	59.7%
51	State Route 51 – 135,849 AADT	16,995	59.7%

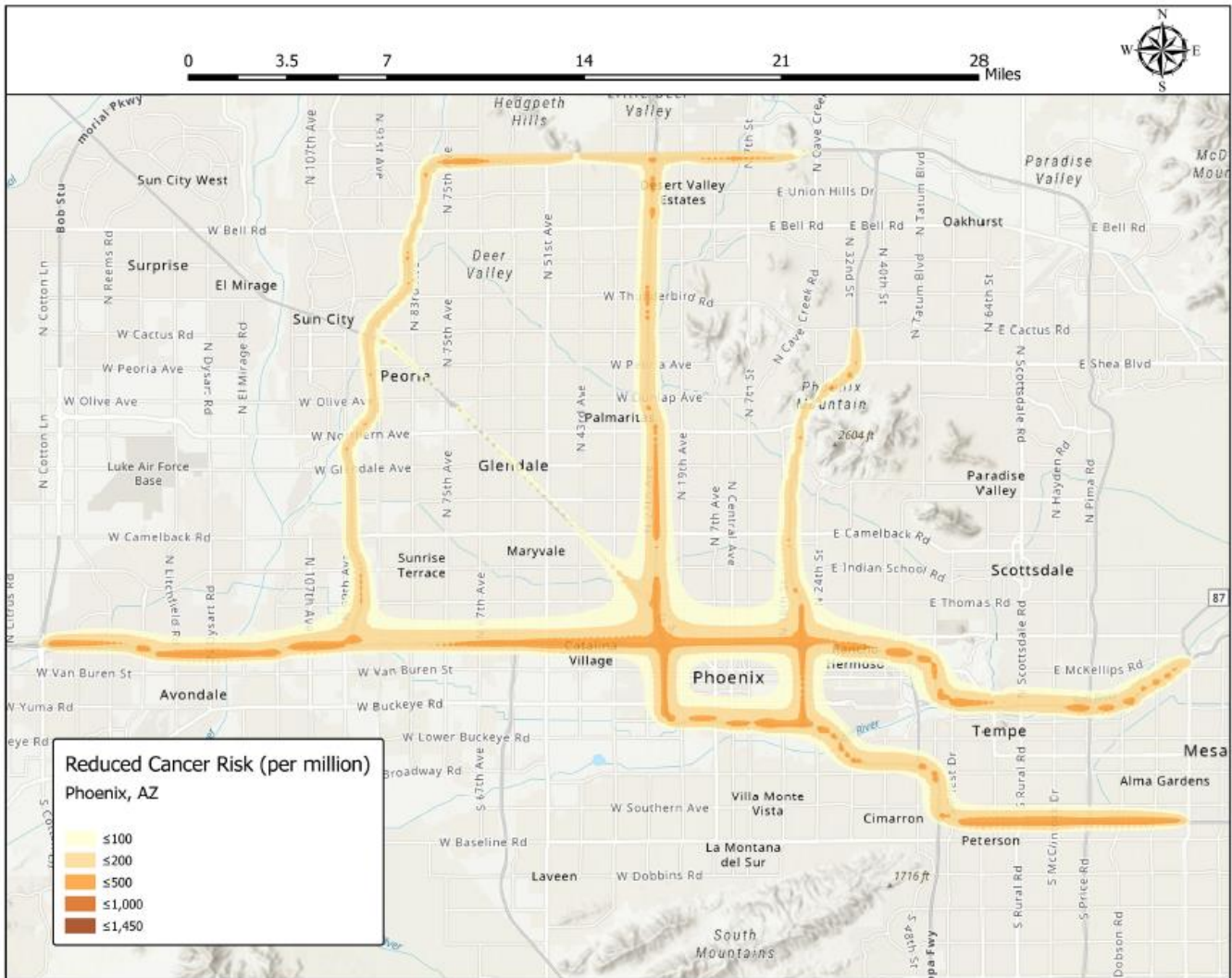
These sources were modeled with unit emission rates in AERMOD, and the Table 6-3 listed emission rates were input into CARB's HARP software to determine cancer risks from the DPM concentrations determined by AERMOD. While dispersion characteristics remained the same between baseline and reduced modeling scenarios, emission rates were reduced according to the number of "old technology" engines combusting diesel, based on source type. The table above shows the Proportion of "Old Technology" Engine Emissions where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

Figure 6-15. Phoenix Baseline Health Risk Assessment Isopleths



The site-specific HRA shows that the PMI is higher than the NATA/HARP evaluation, with an impact of 1,449 cancer cases per million residents. This PMI does not occur at a residential receptor, though, and does not represent an actual risk to residences in the area. The MEIR occurs at 395,744.9 m E, and 3,703,165.8 m N (NAD 83, UTM Zone 12), with a baseline risk of 1,423 cancer cases per million residents. This MEIR is much higher than the NATA/HARP hybrid risks evaluated for that census tract (04013112700) with a risk of 630 in a million.

Figure 6-16. Phoenix Reduced Health Risk Assessment Risk Isopleths



The reduced cancer risk PMI and MEIR are 584 and 573 in 1 million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 850 in 1 million at the MEIR.

6.2.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-4 below.

Table 6-4. Phoenix Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	108.2	\$3,415,926
Asthma Symptoms - Albuterol use	17,977	\$6,213
ER visits - All Cardiac Outcomes	13.1	\$15,156
ER visits – Respiratory	29.0	\$25,343
HA – All – Respiratory	3.8	\$60,870
HA – Alzheimer's Disease	9.2	\$110,046
HA – Cardio Cerebro- and Peripheral Vascular Disease	4.9	\$76,100
HA – Parkinson's Disease	2.4	\$30,359
HA – Respiratory-2	0.7	\$0
HA – Respiratory-2 HA – All Respiratory	4.5	\$0
Incidence – Asthma	142.5	\$6,363,774
Incidence – Hay Fever/Rhinitis	881.7	\$528,913
Incidence – Lung Cancer	6.6	\$84,588
Incidence – Out of Hospital Cardiac Arrest	0.8	\$27,834
Incidence – Stroke	2.8	\$96,060
Minor Restricted Activity Days	39,664	\$2,759,909
Mortality – All Cause	43.5	\$339,485,946
Work Loss Days	6,739	\$1,101,063
Total		\$354,188,100

6.3 Chicago/Naperville, Illinois

6.3.1 NATA Health Risks

The subsections below review the NATA data available for the Chicago/Naperville community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-17 and Figure 6-18 show the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

Figure 6-19 and Figure 6-20 show those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-21 and Figure 6-22 show the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the community of Chicago/Naperville, Illinois.

Because the NATA analysis utilized EPA specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the Chicago/Naperville community.

6.3.1.1 NATA Risk Data

Figure 6-17. North Chicago/Naperville Baseline NATA Total Cancer Risks

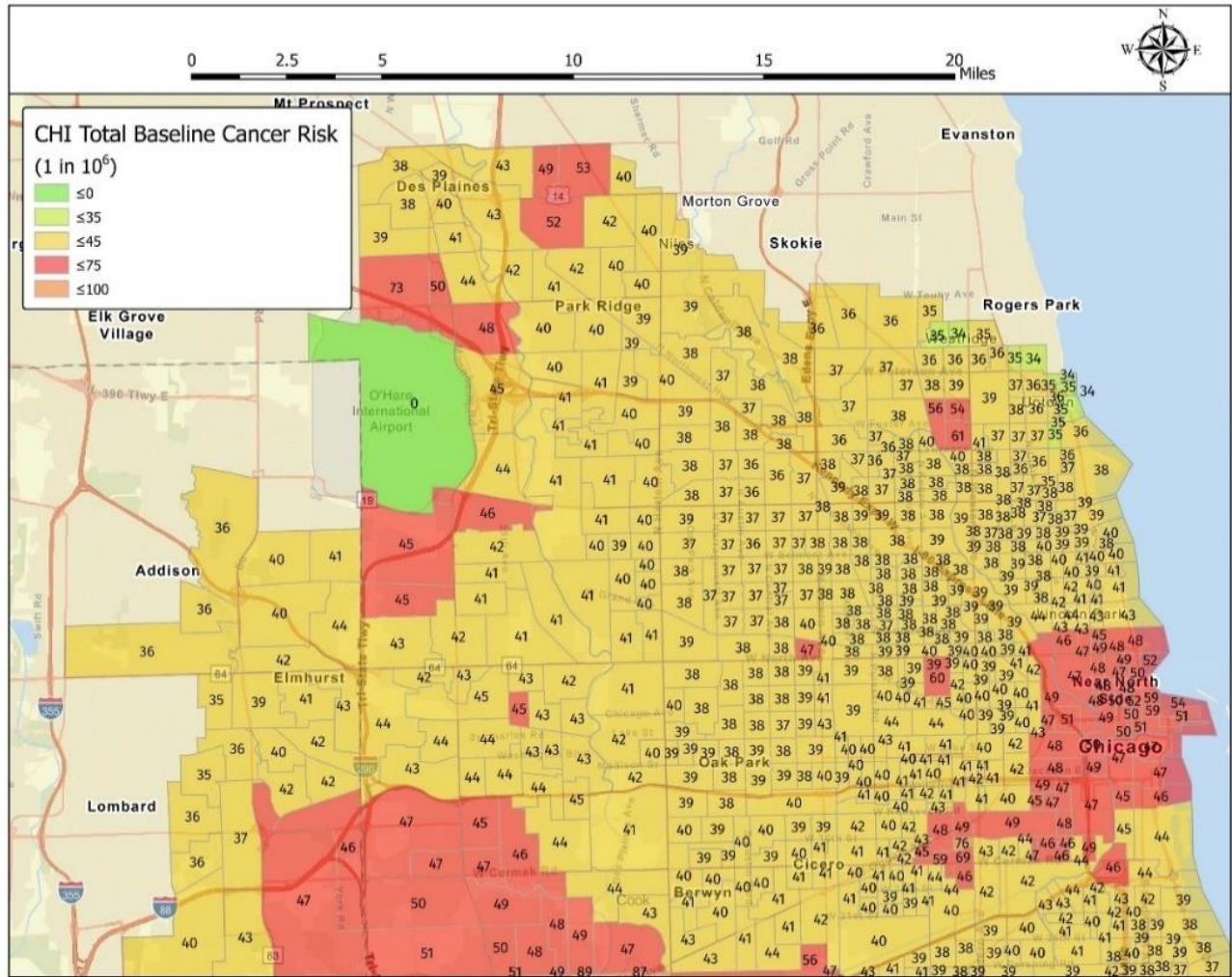
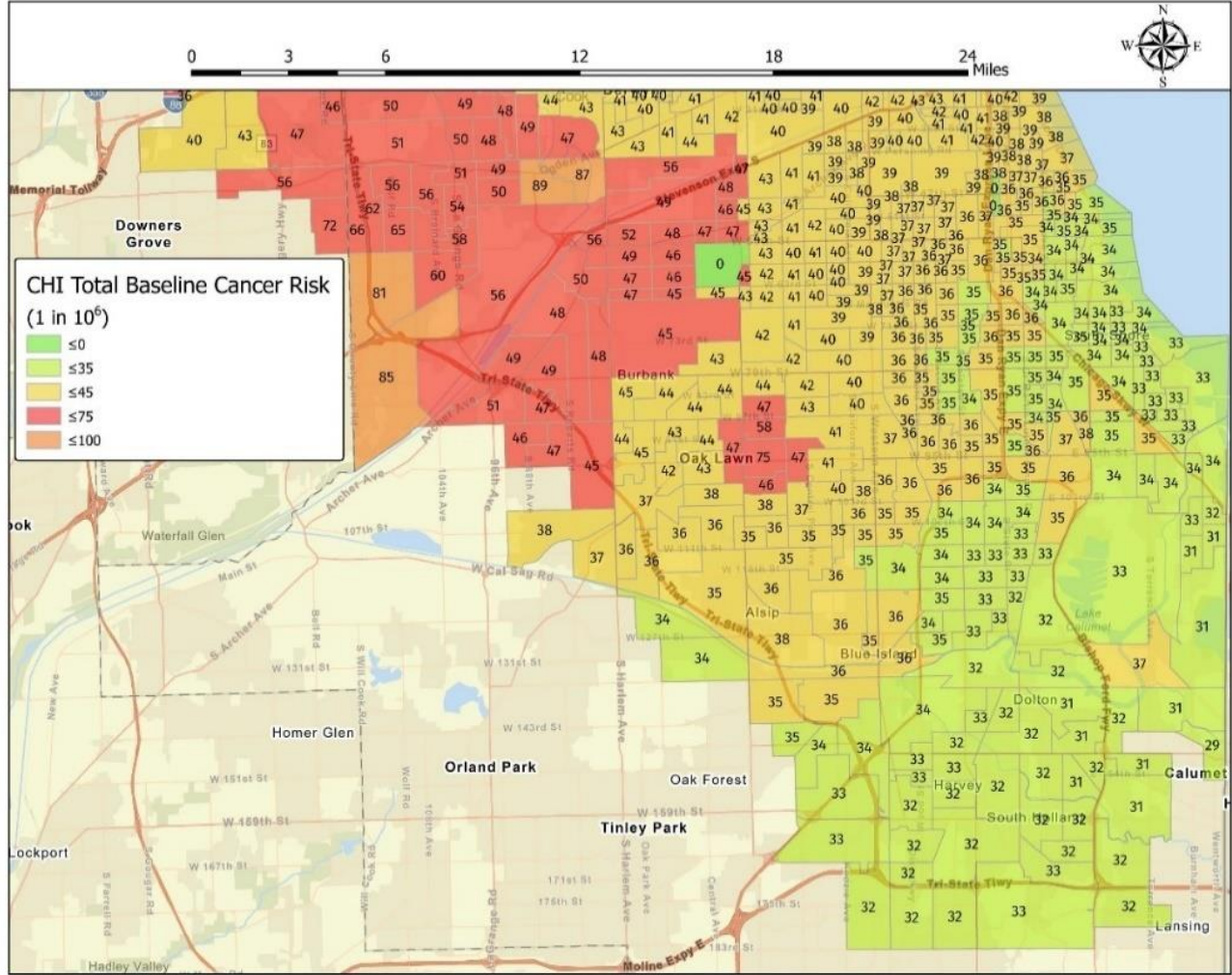


Figure 6-18. South Chicago/Naperville Baseline NATA Total Cancer Risks



According to the NATA, the maximum baseline cancer risk in the Chicago/Naperville community is 89.16 cancer cases per million residents for census tract 17031819200, with a population of 6,107 residents. When accounting for all of the communities assessed, the total cancer burden for the Chicago/Naperville community is 155 cancer cases expected over a 70-year timeline among a total community population of 3,859,930.

Figure 6-19. North Chicago/Naperville Baseline NATA DPM Cancer Risks

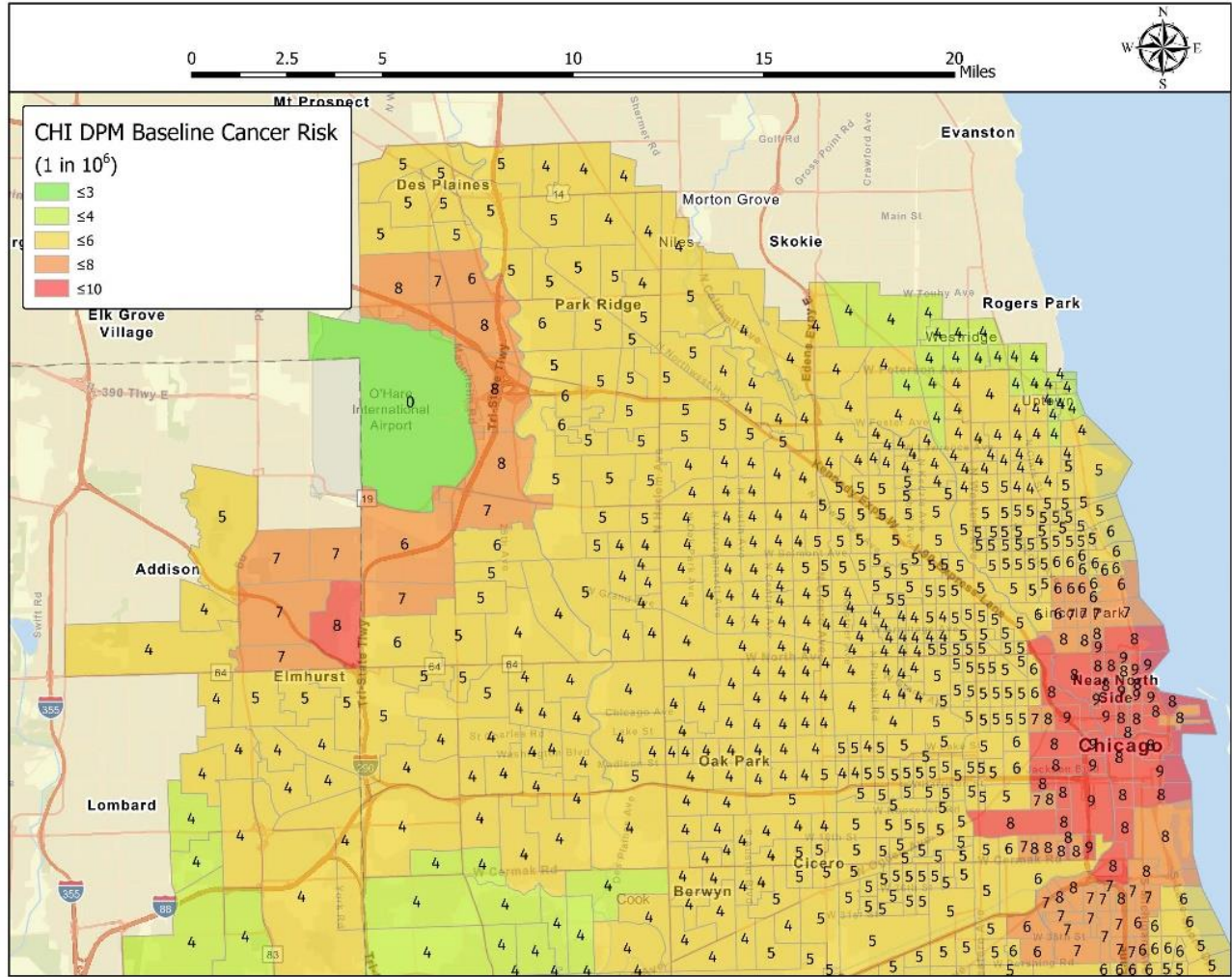
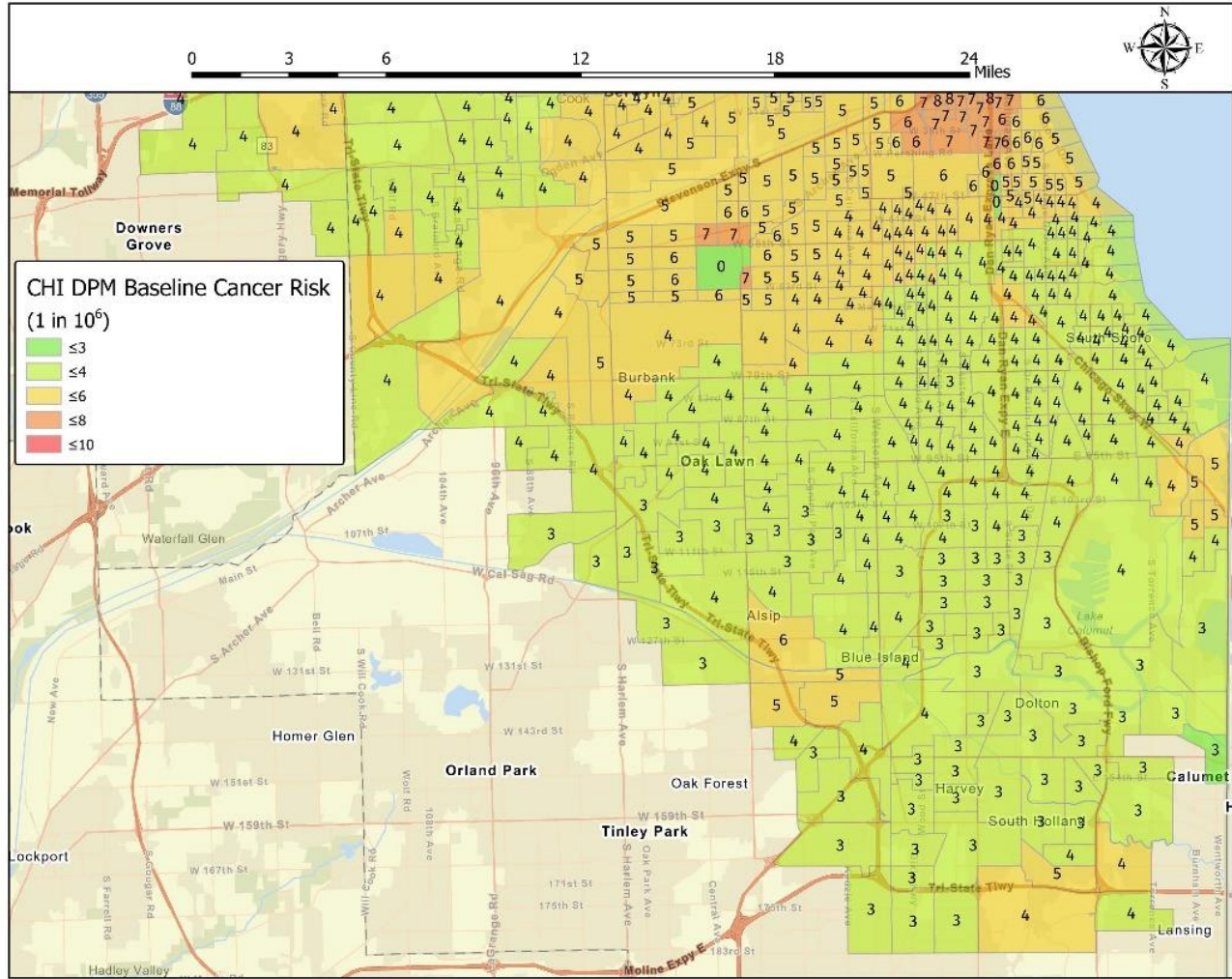


Figure 6-20. South Chicago/Naperville Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the Chicago/Naperville community is 9 cancer cases per million residents for census tract 17031310200, with a population of 1,336 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Chicago/Naperville community is 18 cancer case expected over a 70-year timeline among a total community population of 3,859,930.

Figure 6-21. North Chicago/Naperville Reduced NATA DPM Cancer Risks

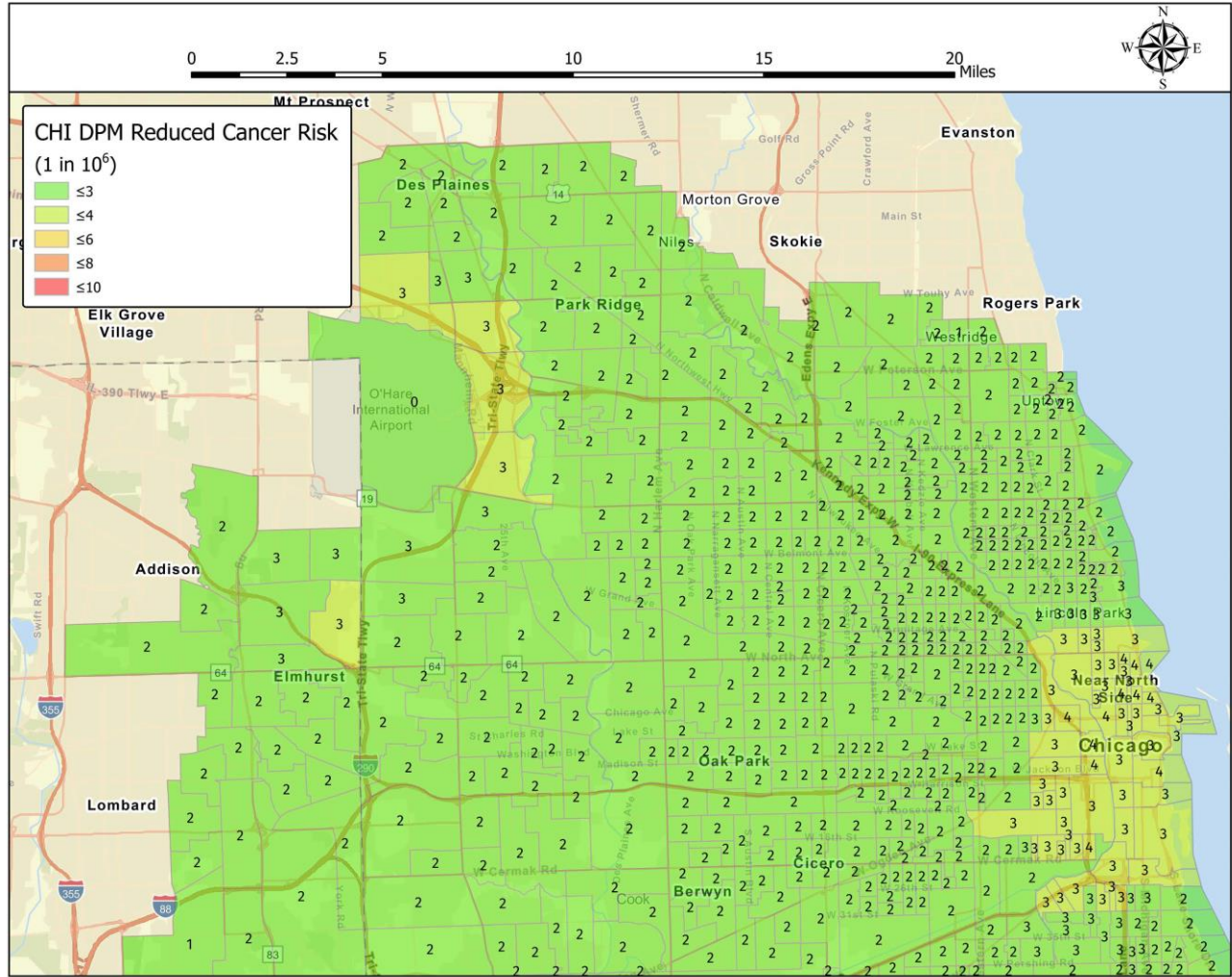
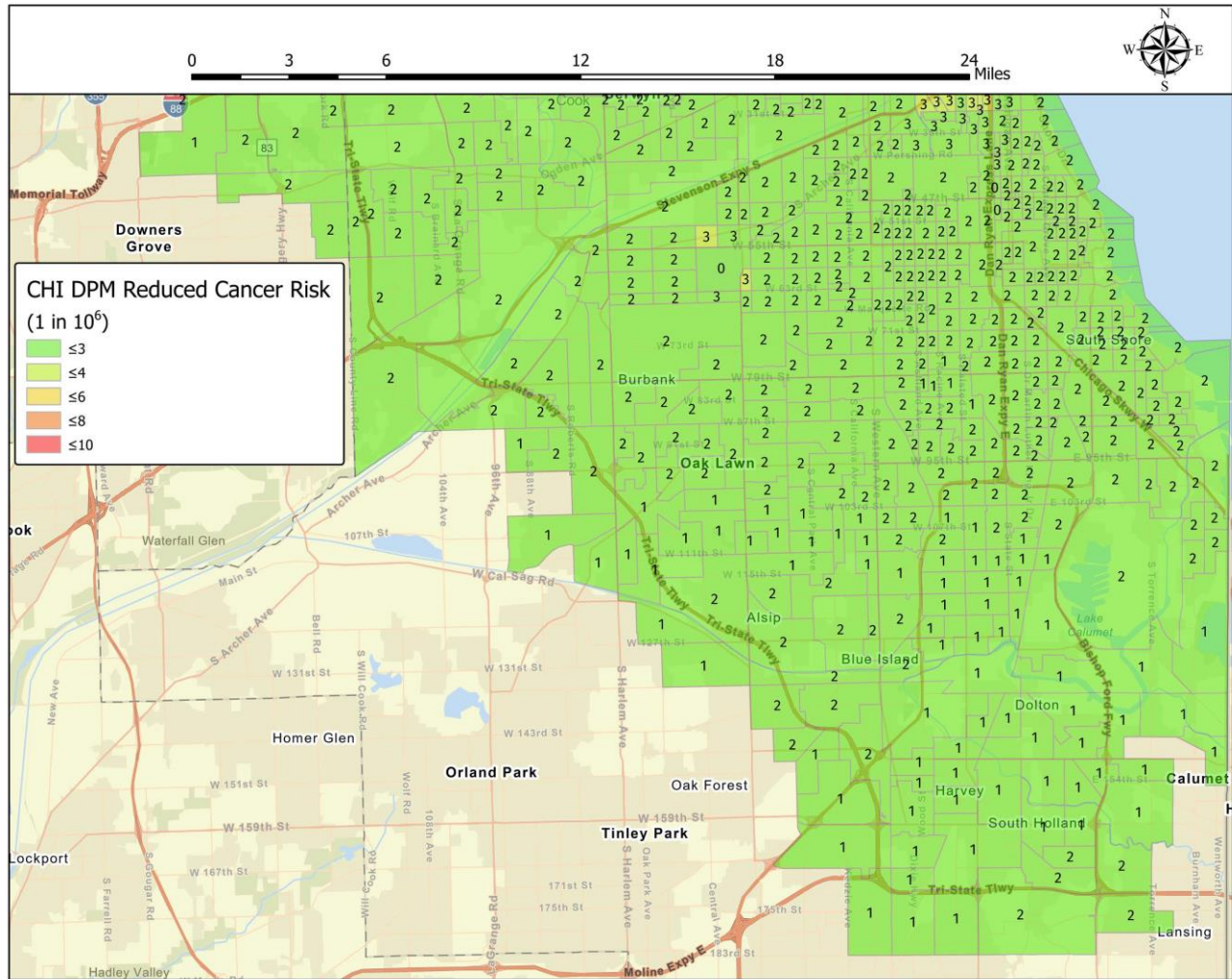


Figure 6-22. South Chicago/Naperville Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Chicago/Naperville community becomes 4 cancer cases per million residents for census tract 17031310200, with a population of 1,336 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Chicago/Naperville community becomes 7 cancer cases expected over a 70-year timeline among a total community population of 3,859,930.

6.3.1.2 NATA Data with HARP Risk Factors.

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB’s HARP program with OEHHA cancer unit risk values.** The data is outlined in the following order:

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program.**

Figure 6-23 and Figure 6-24 shows the baseline DPM concentrations provided by the NATA.

Figure 6-25 and Figure 6-26 shows the baseline DPM-specific cancer risks as determined using the NATA **concentration values and CARB's HARP program.**

Figure 6-27 and Figure 6-28 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Chicago/Naperville community.

Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the Chicago/Naperville community.

Figure 6-23. North Chicago/Naperville Baseline NATA DPM Concentrations

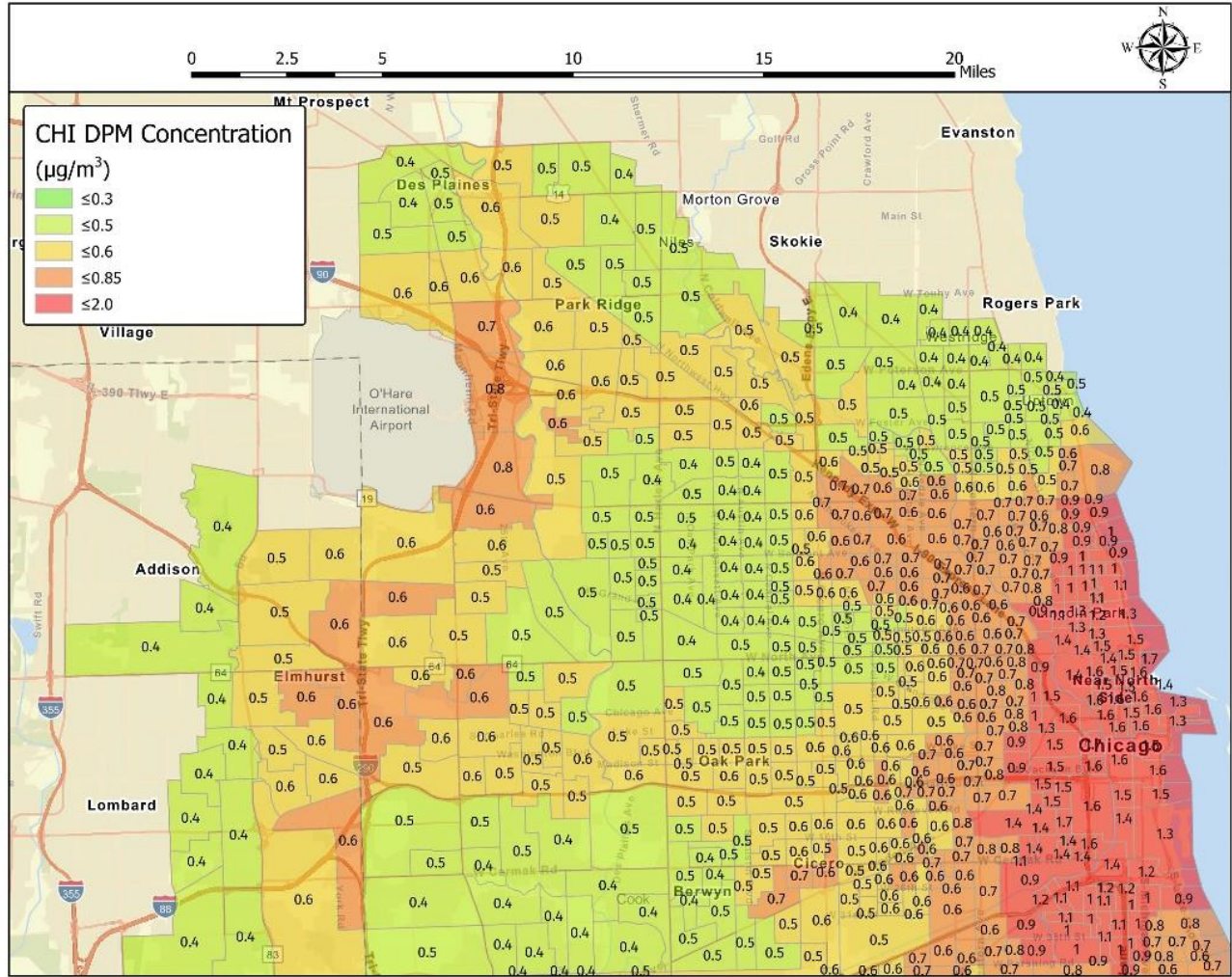
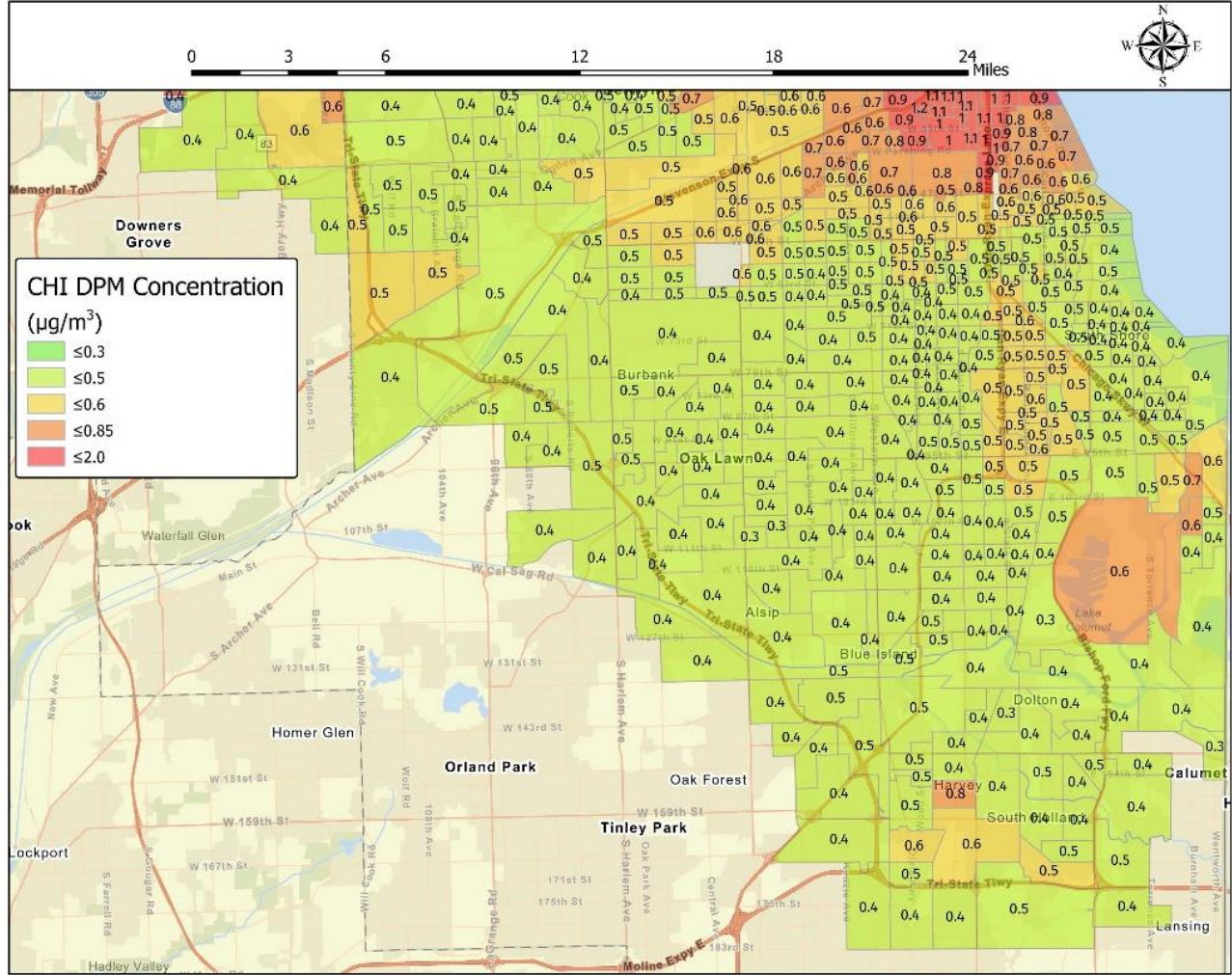


Figure 6-24. South Chicago/Naperville Baseline NATA DPM Concentrations



According to the NATA, the maximum baseline DPM concentration in the Chicago/Naperville community is $1.73 \mu\text{g}/\text{m}^3$ for census tract 17031081202, with a population of 2,995 residents. The average DPM concentration of the Chicago/Naperville community is $0.59 \mu\text{g}/\text{m}^3$.

Figure 6-25. North Chicago/Naperville Baseline NATA/HARP DPM Hybrid Risks

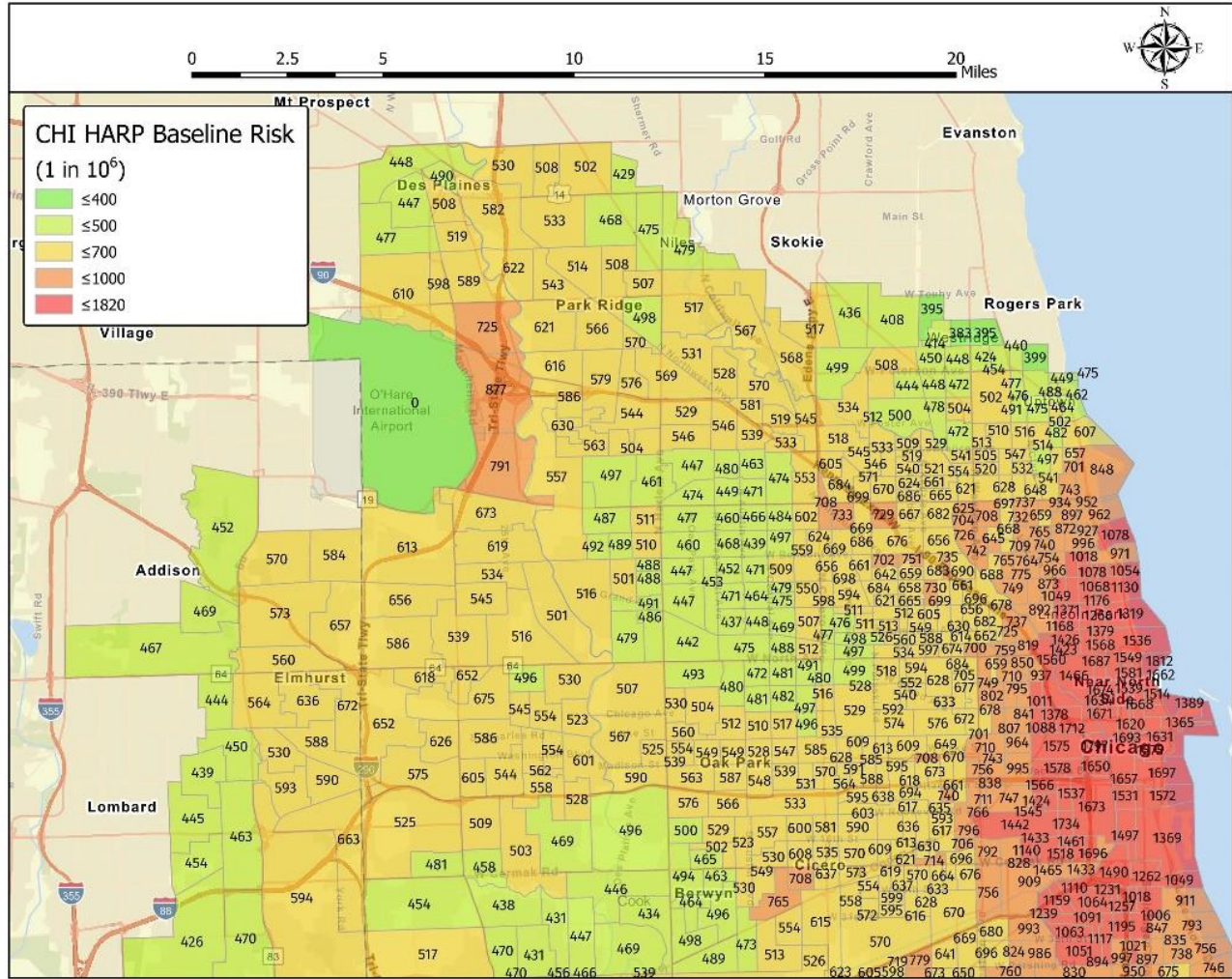
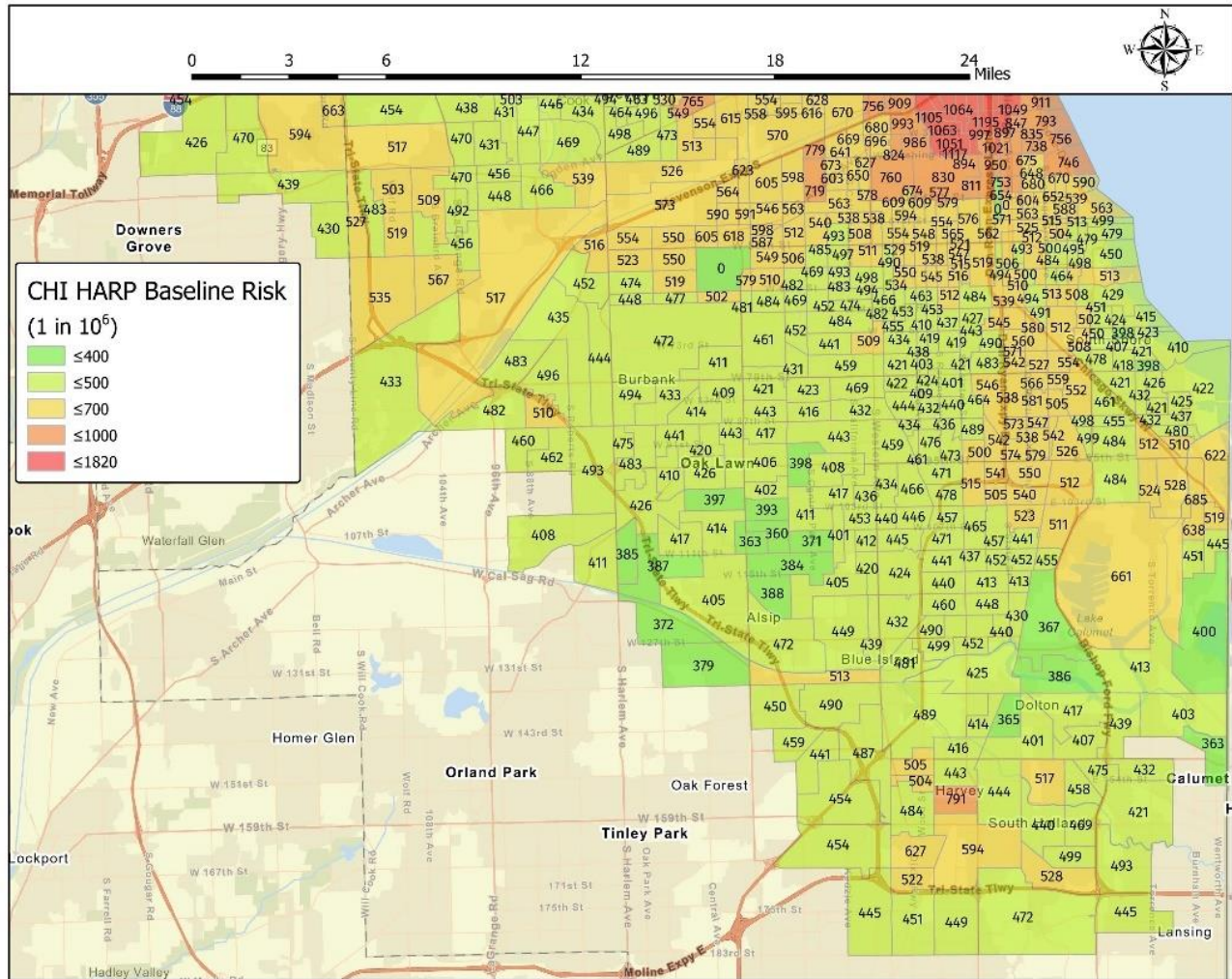


Figure 6-26. South Chicago/Naperville Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the Chicago/Naperville community is 1,812 cancer cases per million residents for census tract 17031081202, with a population of 2,995 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Chicago/Naperville community is 2,361 cancer cases expected over a 70-year timeline among a total community population of 3,859,930.

Figure 6-27. North Chicago/Naperville Reduced NATA/HARP DPM Hybrid Risks

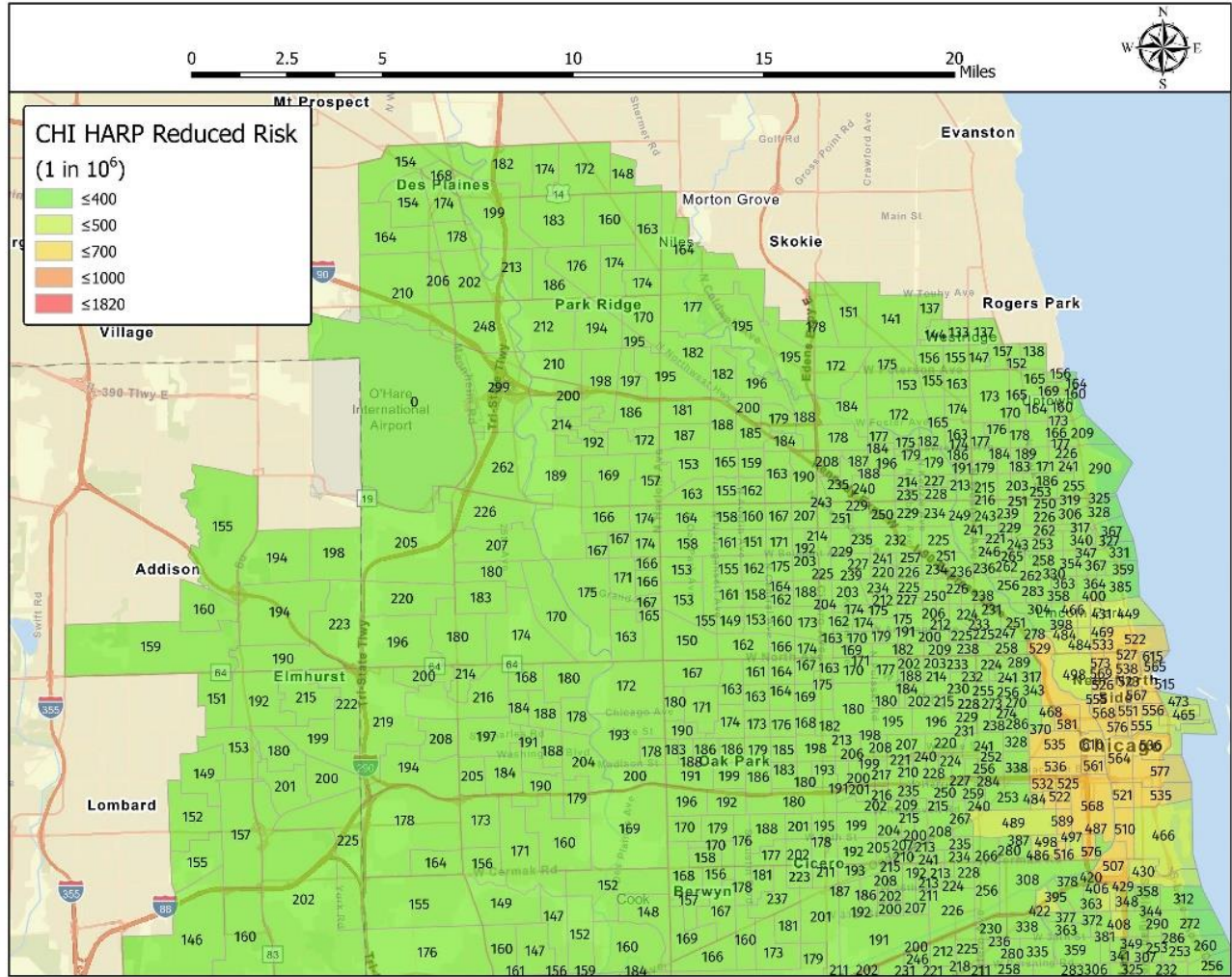
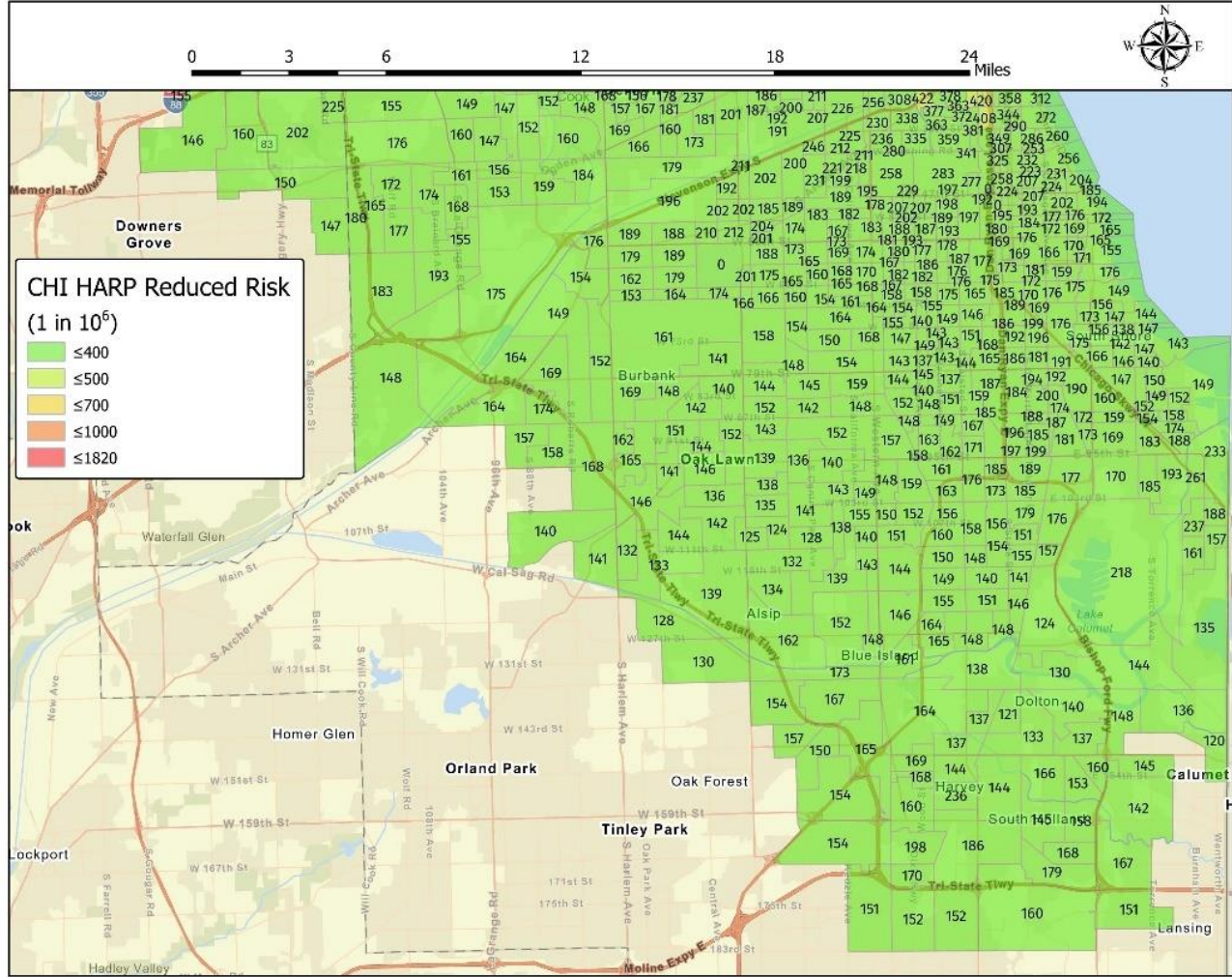


Figure 6-28. South Chicago/Naperville Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Chicago/Naperville community becomes 615 cancer cases per million residents for census tract 17031081202, with a population of 2,995 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Chicago/Naperville community becomes 804 cancer cases expected over a 70-year timeline among a total community population of 3,859,930.

6.3.2 Chicago/Naperville Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Chicago/Naperville. The following sources were utilized to generate the HRA.

- Illinois Department of Transportation (IDOT) (November 4, 2020) – Traffic Counts (2019 Average Annual Daily Traffic)¹⁴

¹⁴ <http://www.sbcity.org/civicax/filebank/blobdload.aspx?blobid=29075>

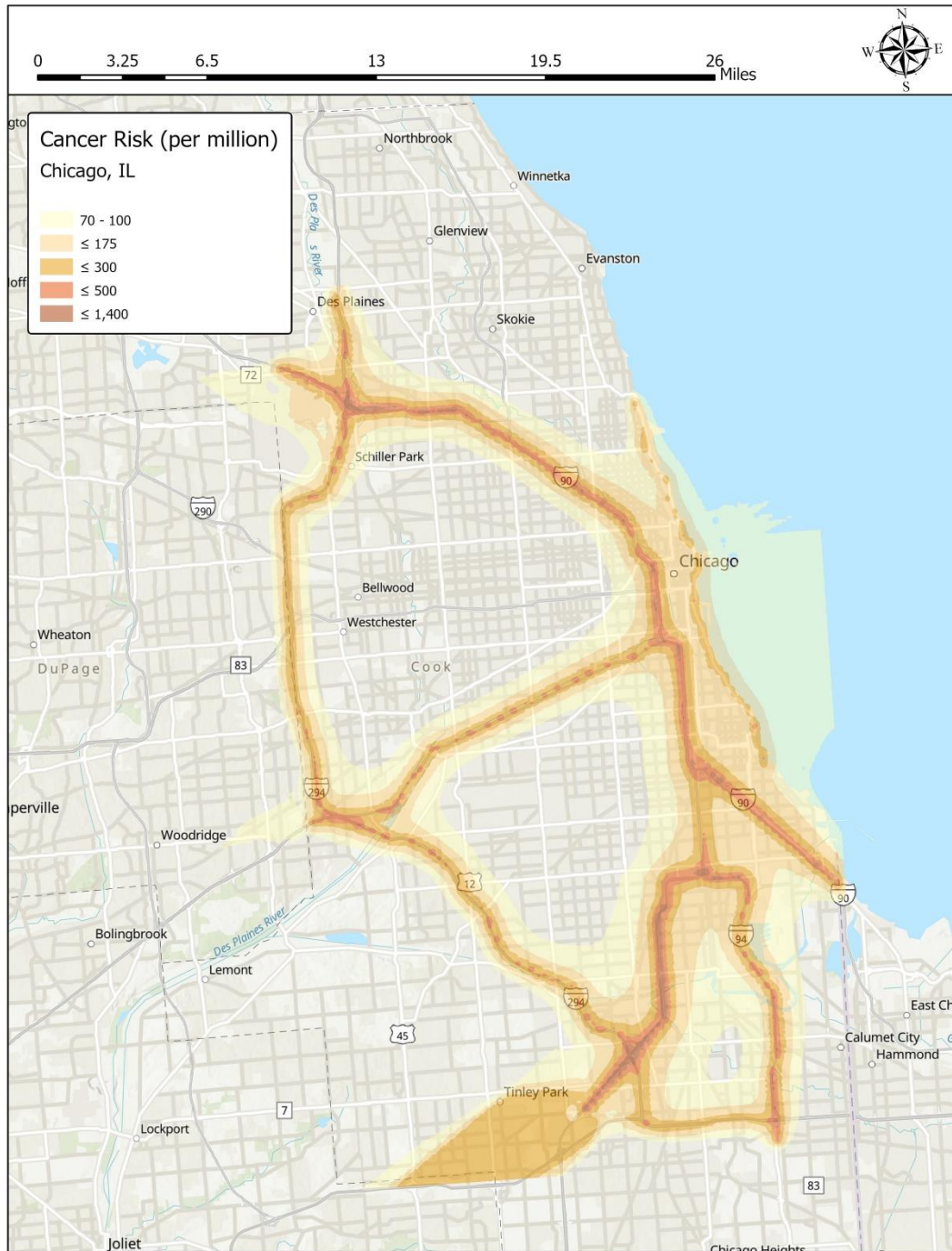
The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-5.

Table 6-5. Chicago/Naperville Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
I-94B	I-94 Business – 166,088 AADT	27,914	59.7%
I-90/EXP	I-90/I-90 Expressway – 220,912 AADT	72,979	59.7%
I-80	I-80 – 136,167 AADT	8,036	59.7%
I-294	I-294 – 161,155 AADT	64,839	59.7%
I-55	I-55 – 148,738 AADT	23,782	59.7%
I-57	I-57 – 266,063 AADT	31,354	59.7%
Lakeshore	Lakeshore Drive – 83,267 AADT	13,308	59.7%

These sources were modeled with unit emission rates in AERMOD, and the Table 6-5 listed emission rates **were input into CARB's HARP software to determine cancer risks from the DPM concentrations determined** by AERMOD. While dispersion characteristics remained the same between baseline and reduced modeling scenarios, emission rates were **reduced according to the number of "old technology" engines combusting diesel**, based on source type. The table above shows the **Proportion of "Old Technology" Engine Emissions** where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

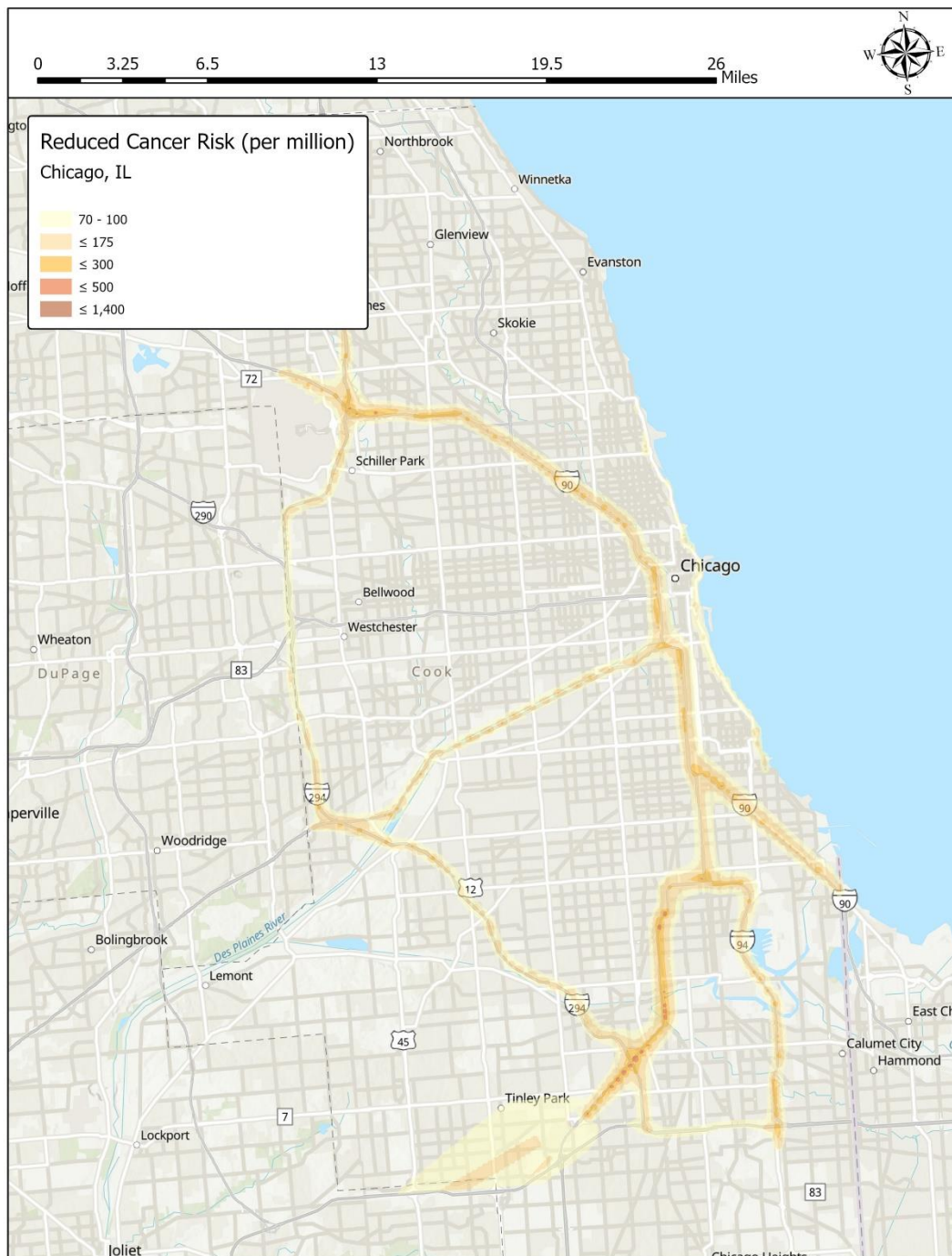
Figure 6-29. Chicago/Naperville Baseline Health Risk Assessment Isopleths



The site-specific HRA shows that the point of maximum impact (PMI) is slightly lower than the NATA/HARP evaluation, with an impact of 1,245 cancer cases per million residents occurs at 445,459.3 m E, and 4,616,593 m N (NAD 83, UTM Zone 16). This PMI occurs at a residential receptor, and therefore represents an actual risk to residences in the area. This PMI/MEIR is lower than the NATA/HARP hybrid risks evaluated for that census tract (17031750100) with a total risk of 471 in a million. This HRA does not capture all of the

cancer-causing sources in the area, but does demonstrate that NATA values are in-line with the site-specific demonstration.

Figure 6-30. Chicago/Naperville Reduced Health Risk Assessment Risk Isoleths



The reduced cancer risk PMI and MEIR are 502 in one million, in the same location as the baseline risk plots. This represents a risk reduction of 743 in 1 million.

6.3.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-6 below.

Table 6-6. Chicago/Naperville Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	259.7	\$8,313,207
Asthma Symptoms - Albuterol use	31,424	\$10,860
ER visits - All Cardiac Outcomes	25.3	\$29,379
ER visits – Respiratory	62.9	\$54,987
HA – All – Respiratory	7.9	\$143,666
HA – Alzheimer’s Disease	40.1	\$490,013
HA – Cardio Cerebro- and Peripheral Vascular Disease	11.4	\$178,295
HA – Parkinson’s Disease	5.4	\$70,206
HA – Respiratory-2	1.8	\$0
HA – Respiratory-2 HA – All Respiratory	9.7	\$0
Incidence – Asthma	240.6	\$10,743,182
Incidence – Hay Fever/Rhinitis	1,511	\$906,354
Incidence – Lung Cancer	11.7	\$146,953
Incidence – Out of Hospital Cardiac Arrest	1.4	\$49,519
Incidence – Stroke	4.7	\$159,847
Minor Restricted Activity Days	73,708	\$5,128,760
Mortality – All Cause	83.0	\$648,242,809
Work Loss Days	12,563	\$2,392,329
Total		\$677,060,365

6.4 Indianapolis, Indiana

6.4.1 NATA Health Risks

The subsections below review the NATA data available for the Indianapolis, IN (Indianapolis) community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-31 shows the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

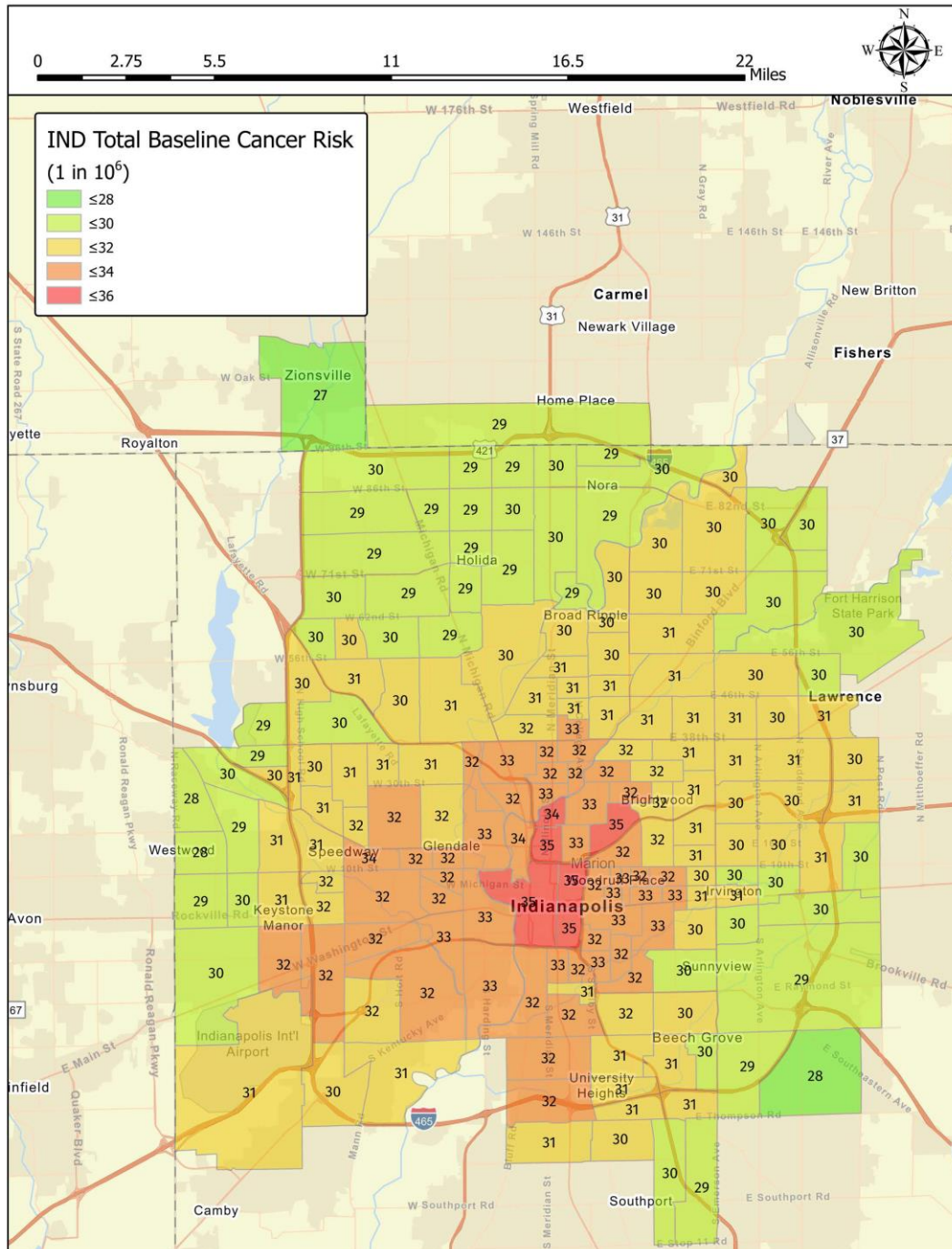
Figure 6-32 shows those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-33 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Indianapolis community.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the Indianapolis community.

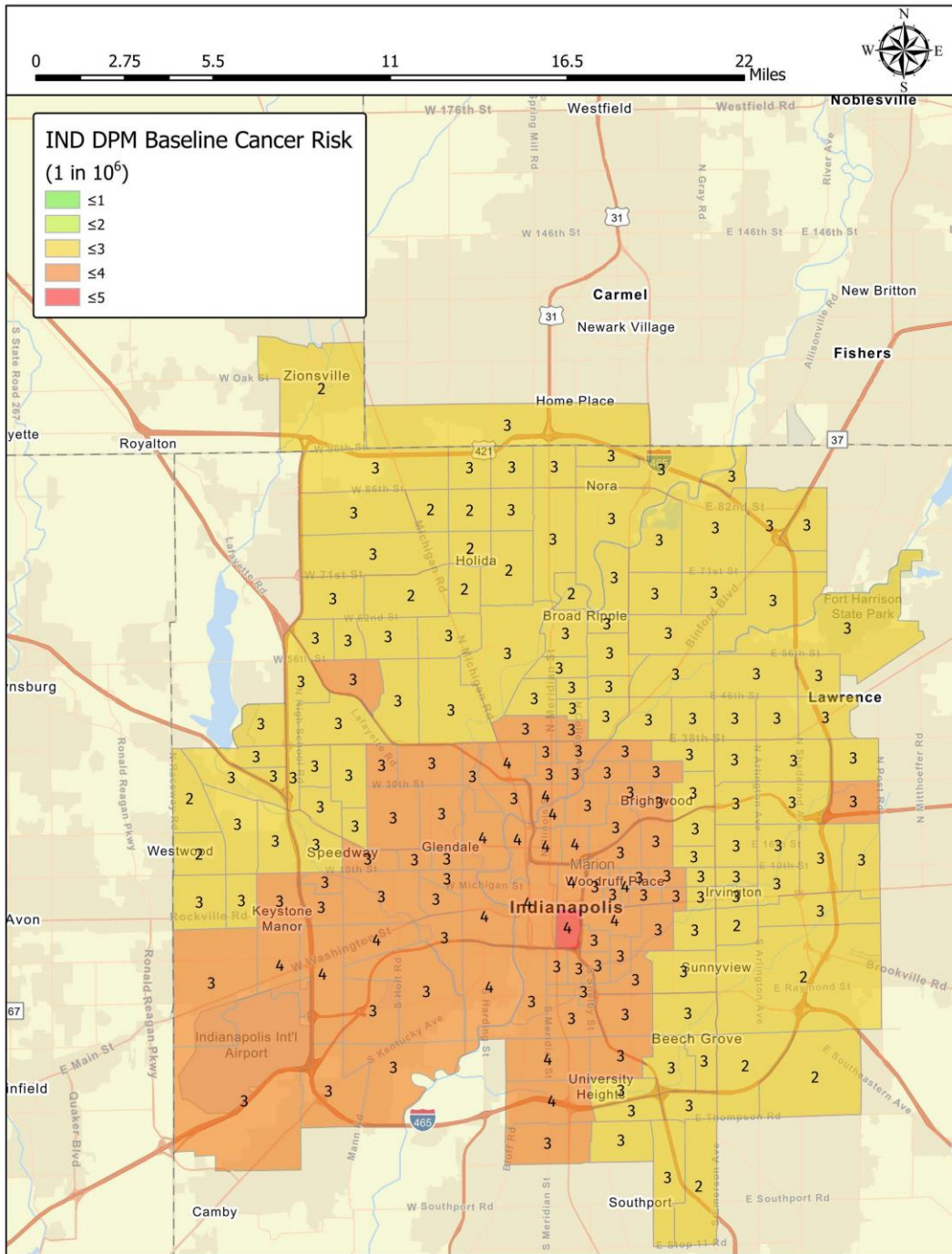
6.4.1.1 NATA Risk Data

Figure 6-31. Indianapolis Baseline NATA Total Cancer Risk



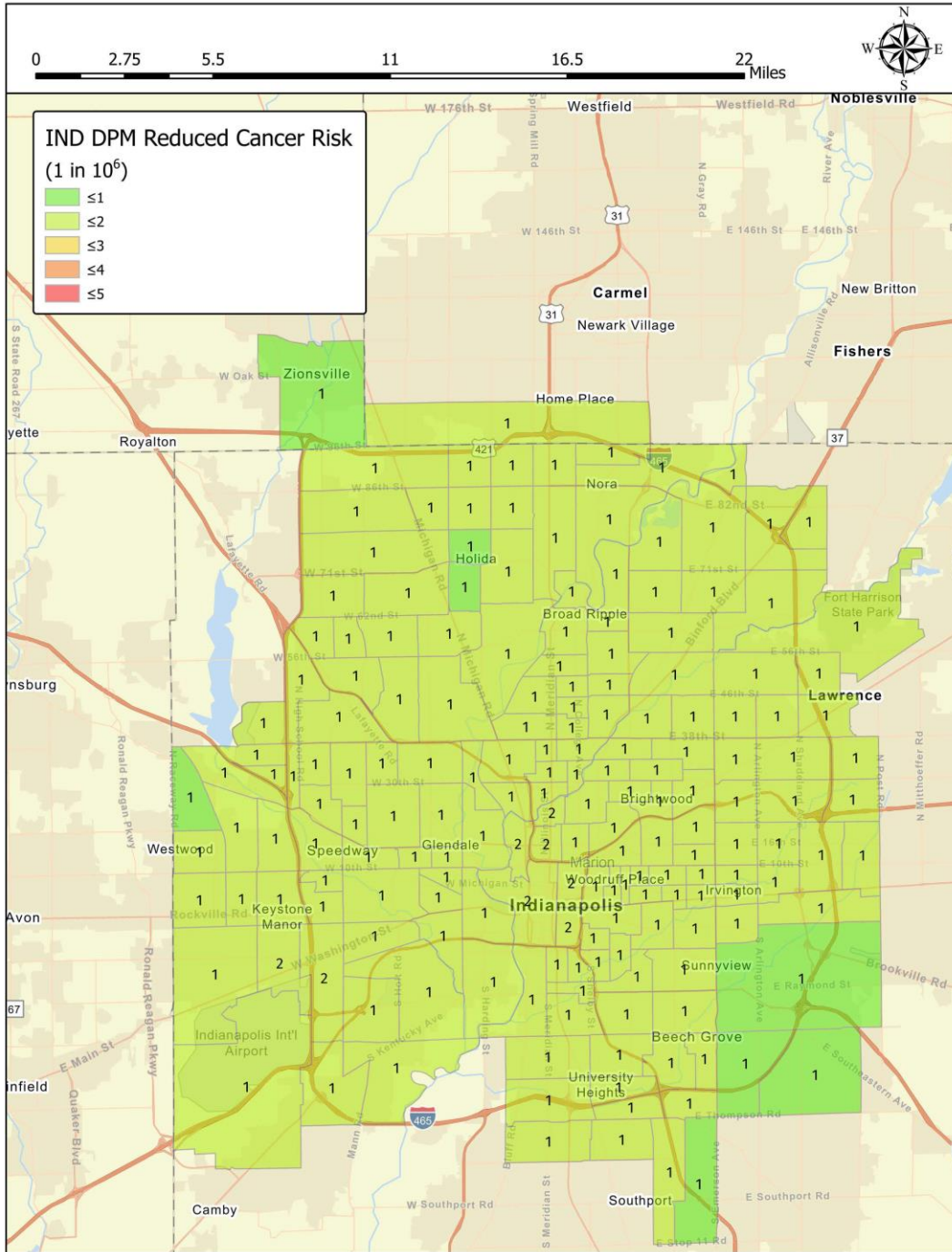
According to the NATA, the maximum baseline cancer risk in the Indianapolis community is 34.92 cancer cases per million residents for census tract 18097353300, with a population of 2,494 residents. When accounting for all of the communities assessed, the total cancer burden for the Indianapolis community is 21 cancer cases expected over a 70-year timeline among a total community population of 672,343.

Figure 6-32. Indianapolis Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the Indianapolis community is 4 cancer cases per million residents for census tract 18097356200, with a population of 2,154 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Indianapolis community is 2 cancer cases expected over a 70-year timeline among a total community population of 672,343.

Figure 6-33. Indianapolis Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Indianapolis community becomes 2 cancer cases per million residents for census tract 18097356200, with a population of 2,154 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Indianapolis community becomes 1 cancer case expected over a 70-year timeline among a total community population of 672,343.

6.4.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB's HARP program with OEHHA cancer unit risk values. The data is outlined in the following order:**

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program.**

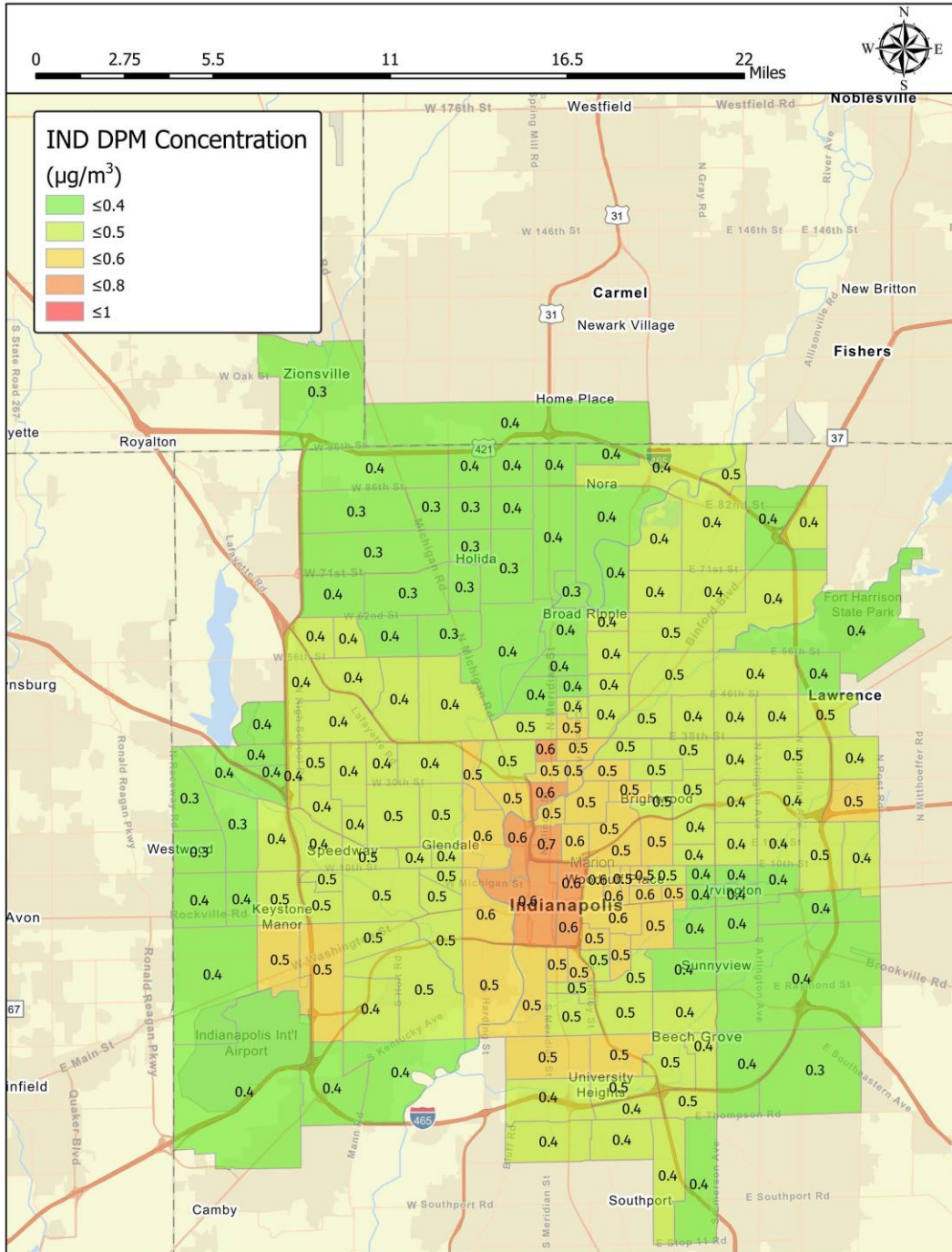
Figure 6-34 shows the baseline DPM concentrations provided by the NATA.

Figure 6-35 shows the baseline DPM-specific cancer risks as determined using the NATA concentration **values and CARB's HARP program.**

Figure 6-36 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Indianapolis community.

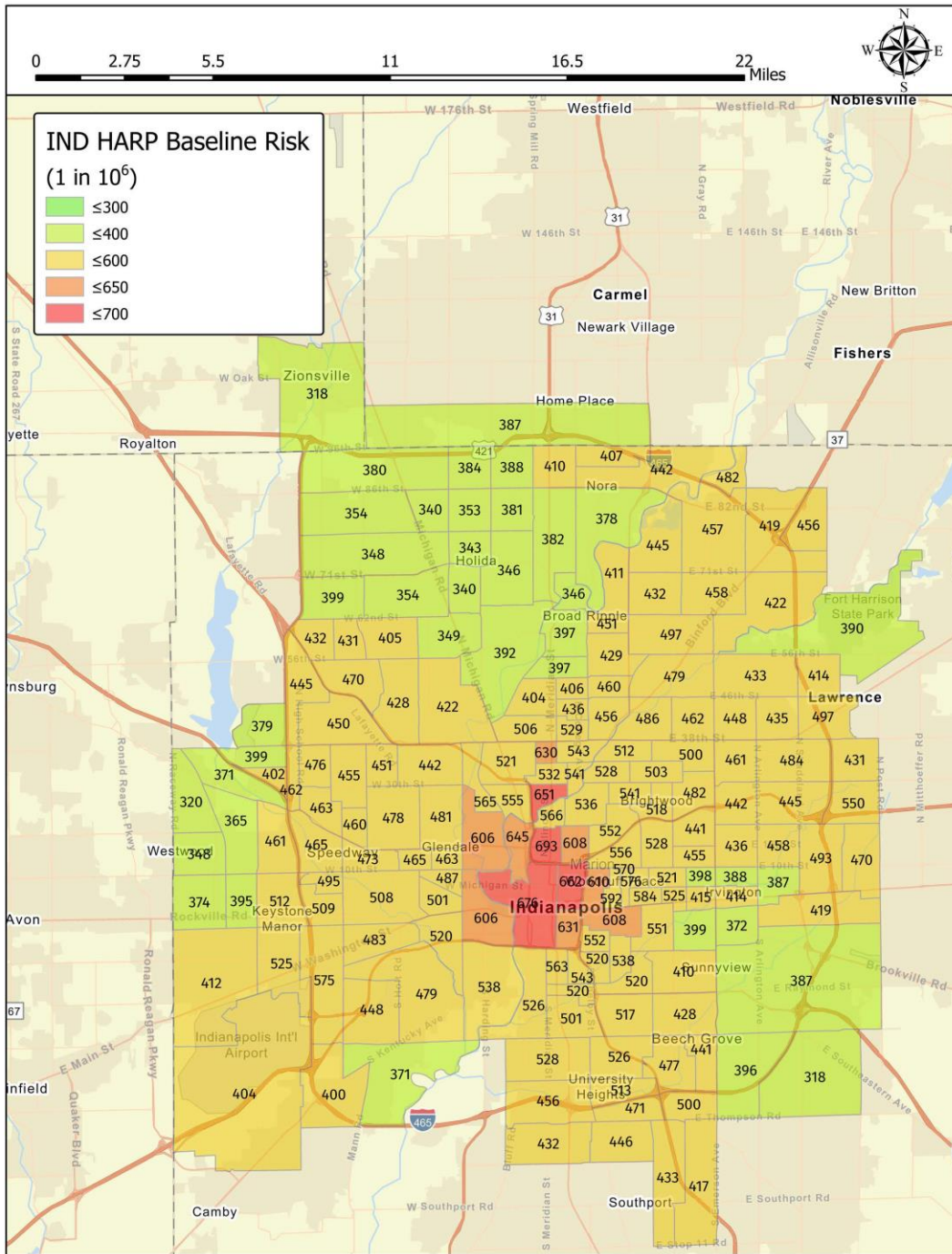
Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the Indianapolis community.

Figure 6-34. Indianapolis Baseline NATA DPM Concentrations



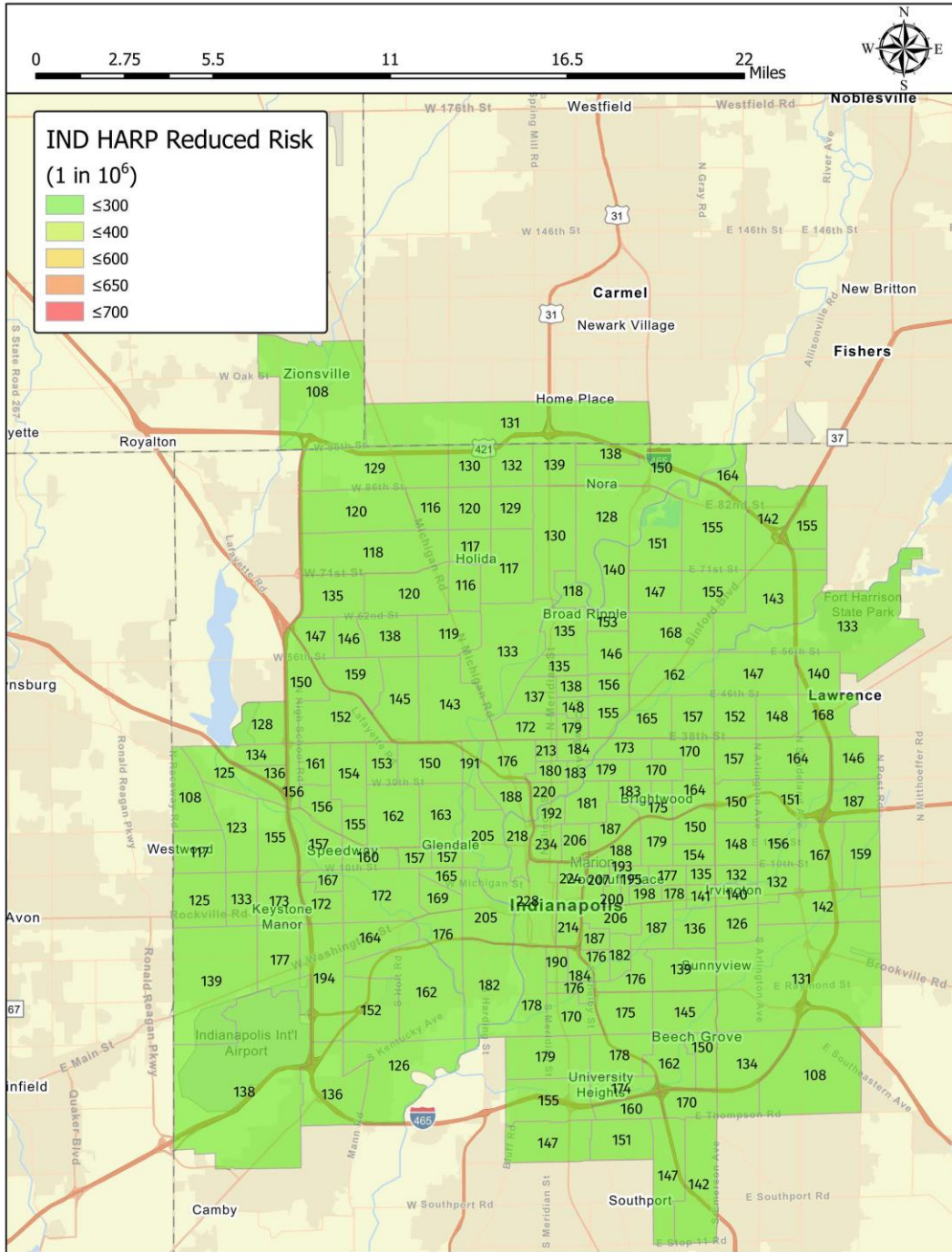
According to the NATA, the maximum baseline DPM concentration in the Indianapolis community is $0.66 \mu\text{g}/\text{m}^3$ for census tract 18097353300, with a population of 2,494 residents. The average DPM concentration of the Indianapolis community is $0.44 \mu\text{g}/\text{m}^3$.

Figure 6-35. Indianapolis Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the Indianapolis community is 693 cancer cases per million residents for census tract 18097353300, with a population of 2,494 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Indianapolis community is 305 cancer cases expected over a 70-year timeline among a total community population of 672,343.

Figure 6-36. Indianapolis Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Indianapolis community becomes 234 cancer cases per million residents for census tract 18097353300, with a population of 2,494 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Indianapolis community becomes 103 cancer cases expected over a 70-year timeline among a total community population of 672,343.

6.4.2 Indianapolis Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Indianapolis. The following sources were utilized to generate the HRA.

- Indiana Department of Transportation (INDOT) – Traffic Counts (Average Annual Daily Traffic)¹⁵

The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-7.

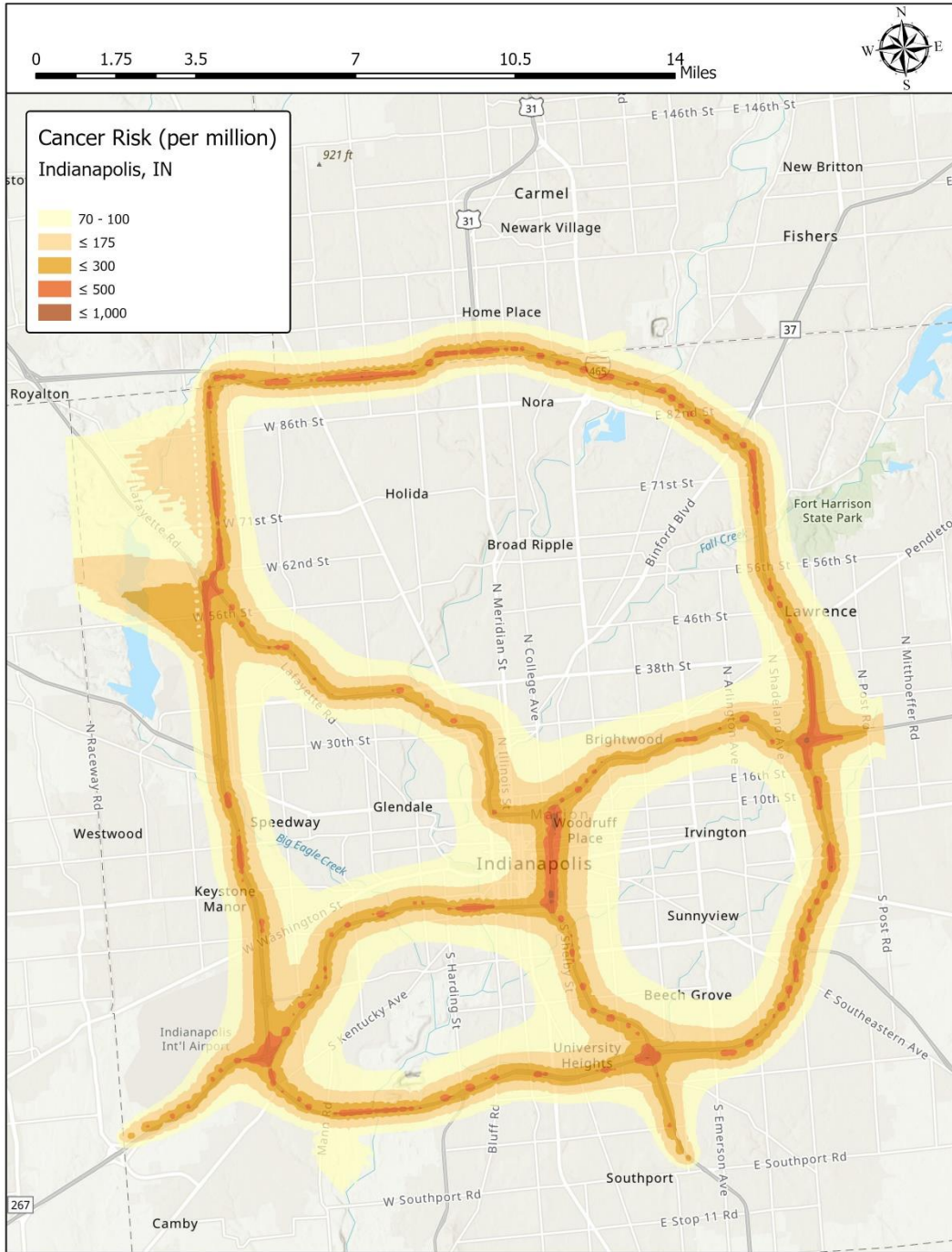
Table 6-7. Indianapolis Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
I-465	I-465 – 124,352 AADT	65,956	59.7%
I-65	I-65 – 99,383 AADT	19,383	59.7%
I-70	I-70 – 98,542 AADT	21,584	59.7%

These sources were modeled with unit emission rates in AERMOD, and the Table 6-7. listed emission rates **were input into CARB's HARP software to determine cancer risks from the DPM concentrations determined by AERMOD.** While dispersion characteristics remained the same between baseline and reduced modeling scenarios, **emission rates were reduced according to the number of "old technology" engines combusting diesel, based on source type.** The table above shows the **Proportion of "Old Technology" Engine Emissions** where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

¹⁵ <https://www.in.gov/indot/about-indot/central-office/asset-data-collection/traffic-statistics/>

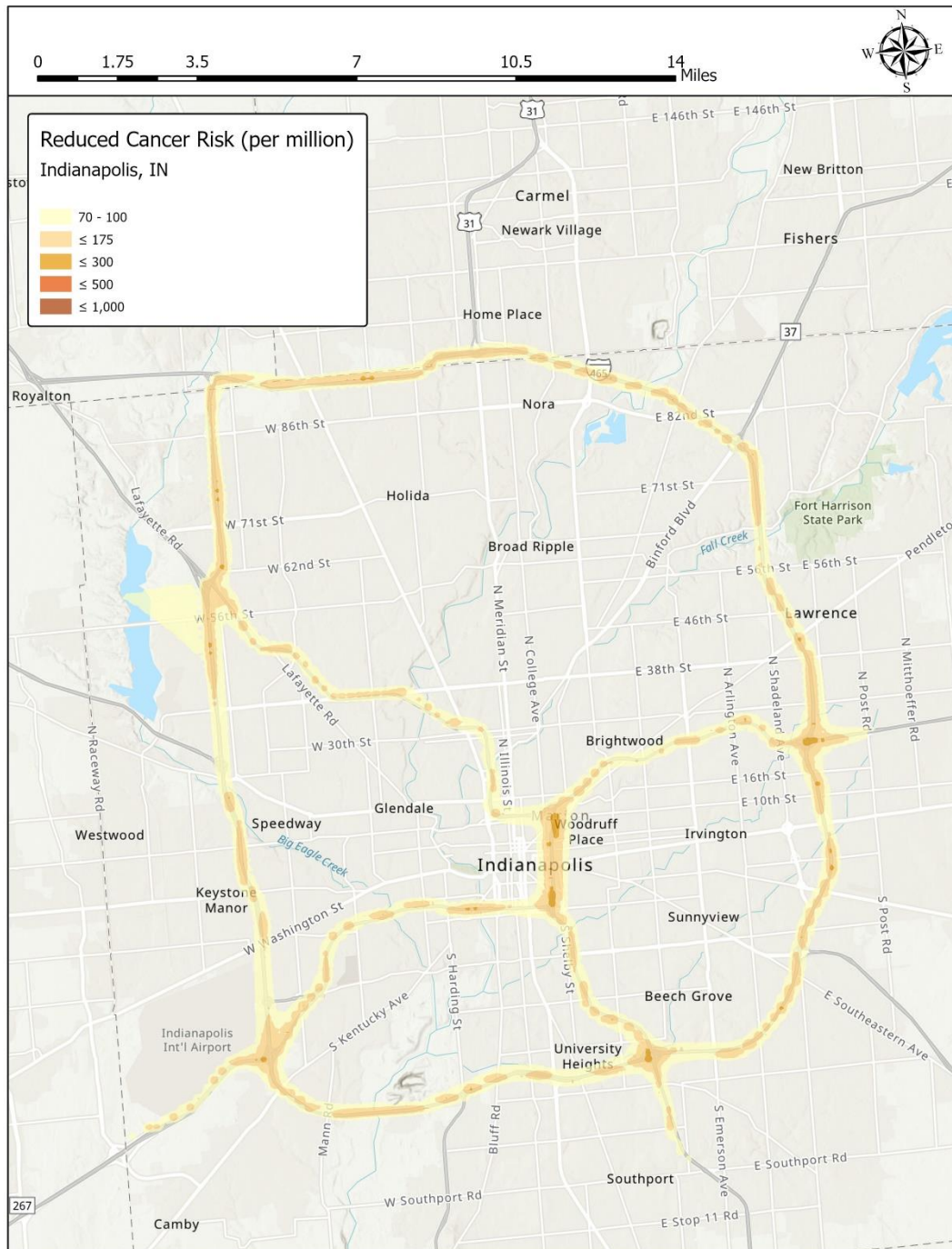
Figure 6-37. Indianapolis Baseline Health Risk Assessment Isoleths



The site-specific HRA shows that the point of maximum impact (PMI) is similar to the NATA/HARP evaluation, with an impact of 790 cancer cases per million residents. This PMI does not occur at a residential receptor, though, and does not represent an actual risk to residences in the area. The MEIR occurs at 582,910.8 m E, and 4,404,413.8 m N (NAD 83, UTM Zone 16), with a baseline risk of 590 cancer cases per million residents. This MEIR is higher than the NATA/HARP hybrid risks evaluated for that census

tract (18097360602) with a total risk of 493 in a million. This HRA does not capture all of the cancer-causing sources in the area, but does demonstrate that NATA values are in-line with the site-specific demonstration.

Figure 6-38. Indianapolis Reduced Health Risk Assessment Isoleths



The reduced cancer risk PMI and MEIR are 318 and 238 in 1 million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 352 in 1 million at the MEIR.

6.4.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-8 below.

Table 6-8. Indianapolis Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	42.9	\$1,405,720
Asthma Symptoms - Albuterol use	6,353	\$2,195
ER visits - All Cardiac Outcomes	4.6	\$5,392
ER visits – Respiratory	0.1	\$125
HA – All – Respiratory	1.6	\$25,980
HA – Alzheimer’s Disease	6.1	\$73,347
HA – Cardio Cerebro- and Peripheral Vascular Disease	1.8	\$27,675
HA – Parkinson’s Disease	0.9	\$11,217
HA – Respiratory-2	0.3	\$0
HA – Respiratory-2 HA – All Respiratory	1.9	\$0
Incidence – Asthma	49.3	\$2,201,336
Incidence – Hay Fever/Rhinitis	304.0	\$182,358
Incidence – Lung Cancer	1.9	\$24,283
Incidence – Out of Hospital Cardiac Arrest	0.2	\$8,347
Incidence – Stroke	0.8	\$25,740
Minor Restricted Activity Days	12,972	\$902,604
Mortality – All Cause	14.4	\$111,705,133
Work Loss Days	2,209	\$374,359
Total		\$116,975,811

6.5 Boston, Massachusetts

6.5.1 NATA Health Risks

The subsections below review the NATA data available for the Boston, MA (Boston) community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-39 shows the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

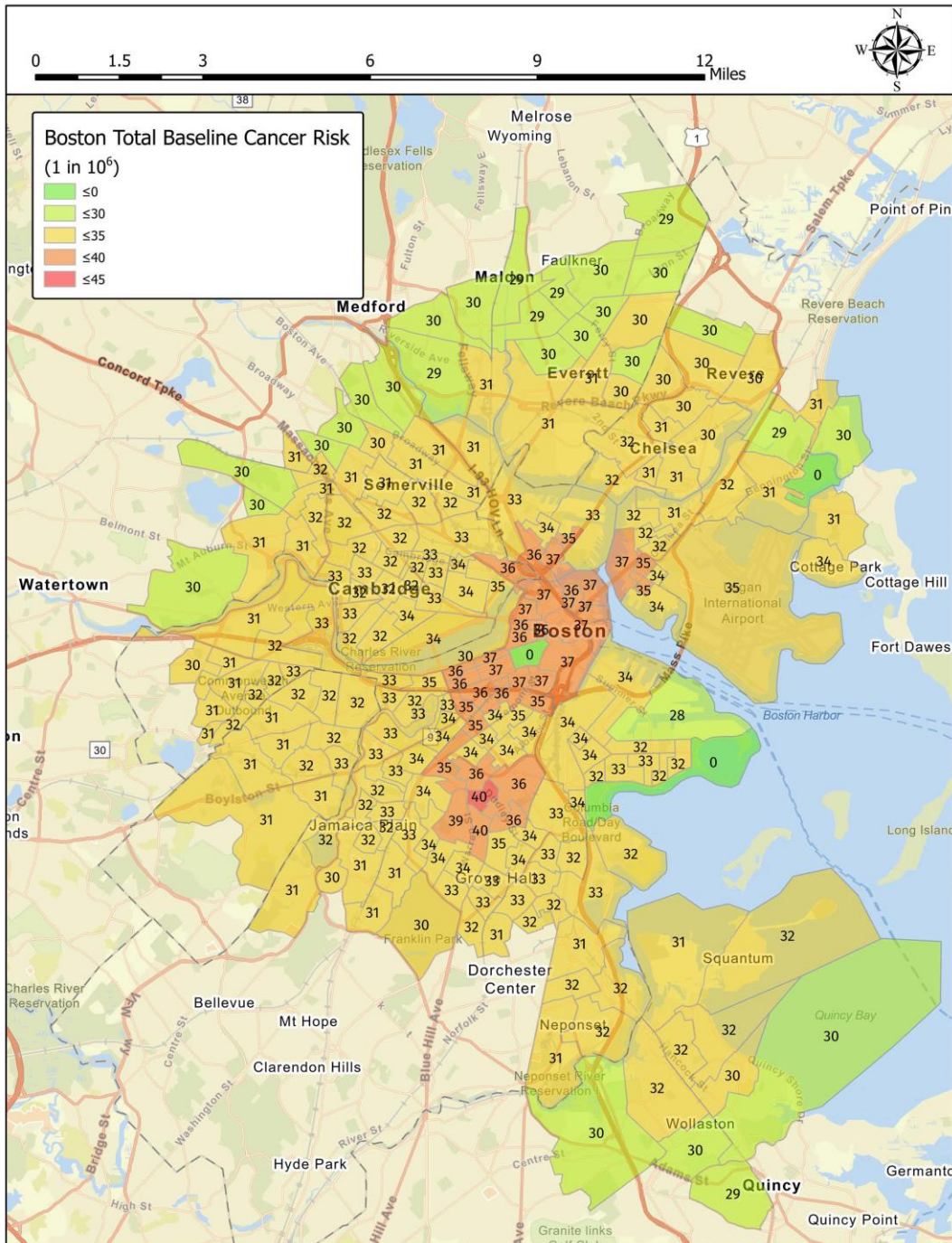
Figure 6-40 shows those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-41 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Boston community.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the Boston community.

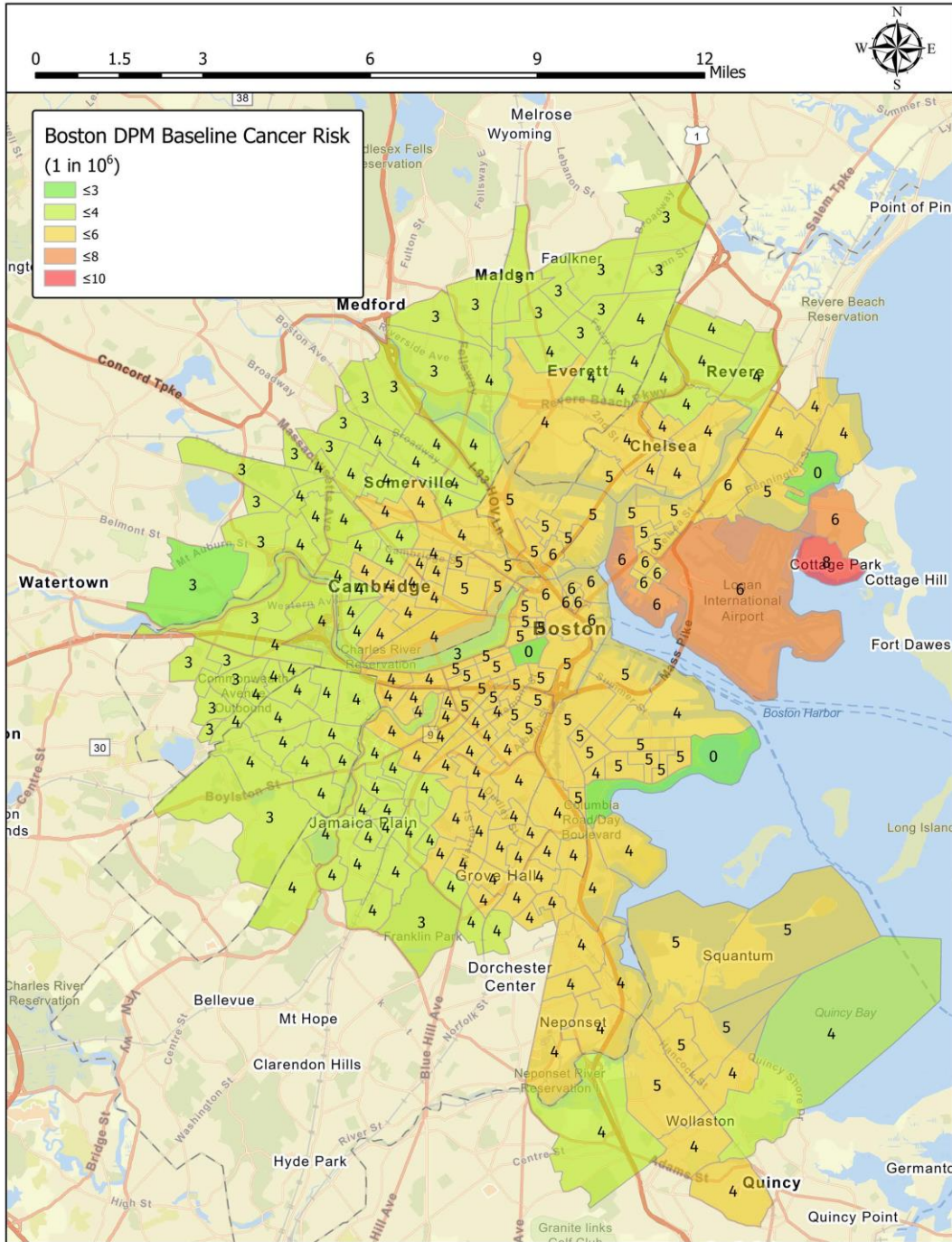
6.5.1.1 NATA Risk Data

Figure 6-39. Boston Baseline NATA Total Cancer Risks



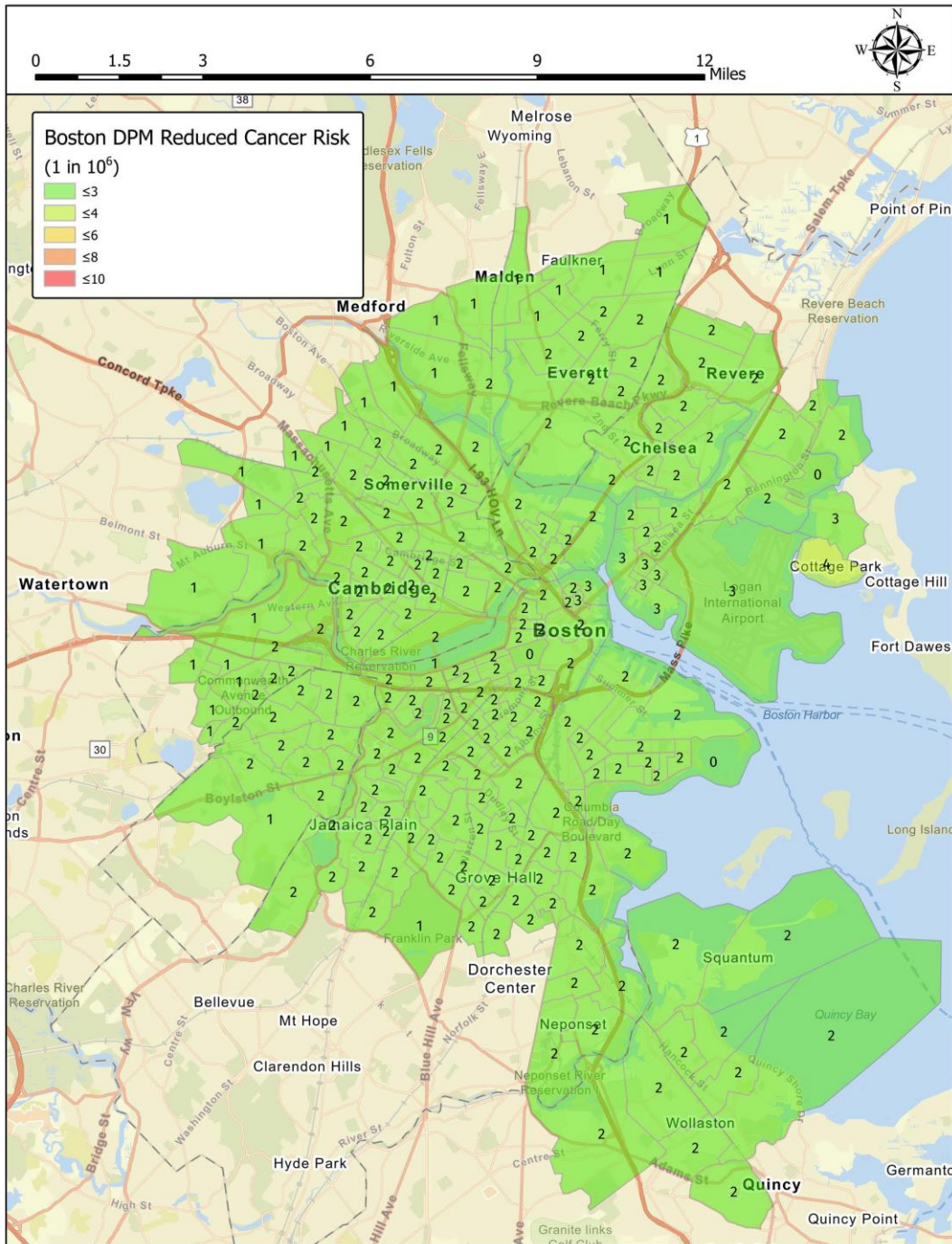
According to the NATA, the maximum baseline cancer risk in the Boston community is 40 cancer cases per million residents for census tract 25025080300, with a population of 1,769 residents. When accounting for all of the communities assessed, the total cancer burden for the Boston community is 28 cancer cases expected over a 70-year timeline among a total community population of 870,752.

Figure 6-40. Boston Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the Boston community is 8 cancer cases per million residents for census tract 25025180301, with a population of 3,024 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Boston community is 4 cancer cases expected over a 70-year timeline among a total community population of 870,752.

Figure 6-41. Boston Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Boston community becomes 4 cancer cases per million residents for census tract 25025180301, with a population of 3,024 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Boston community becomes 2 cancer cases expected over a 70-year timeline among a total community population of 870,752.

6.5.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB's HARP program with OEHHA cancer unit risk values**. The data is outlined in the following order:

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program**.

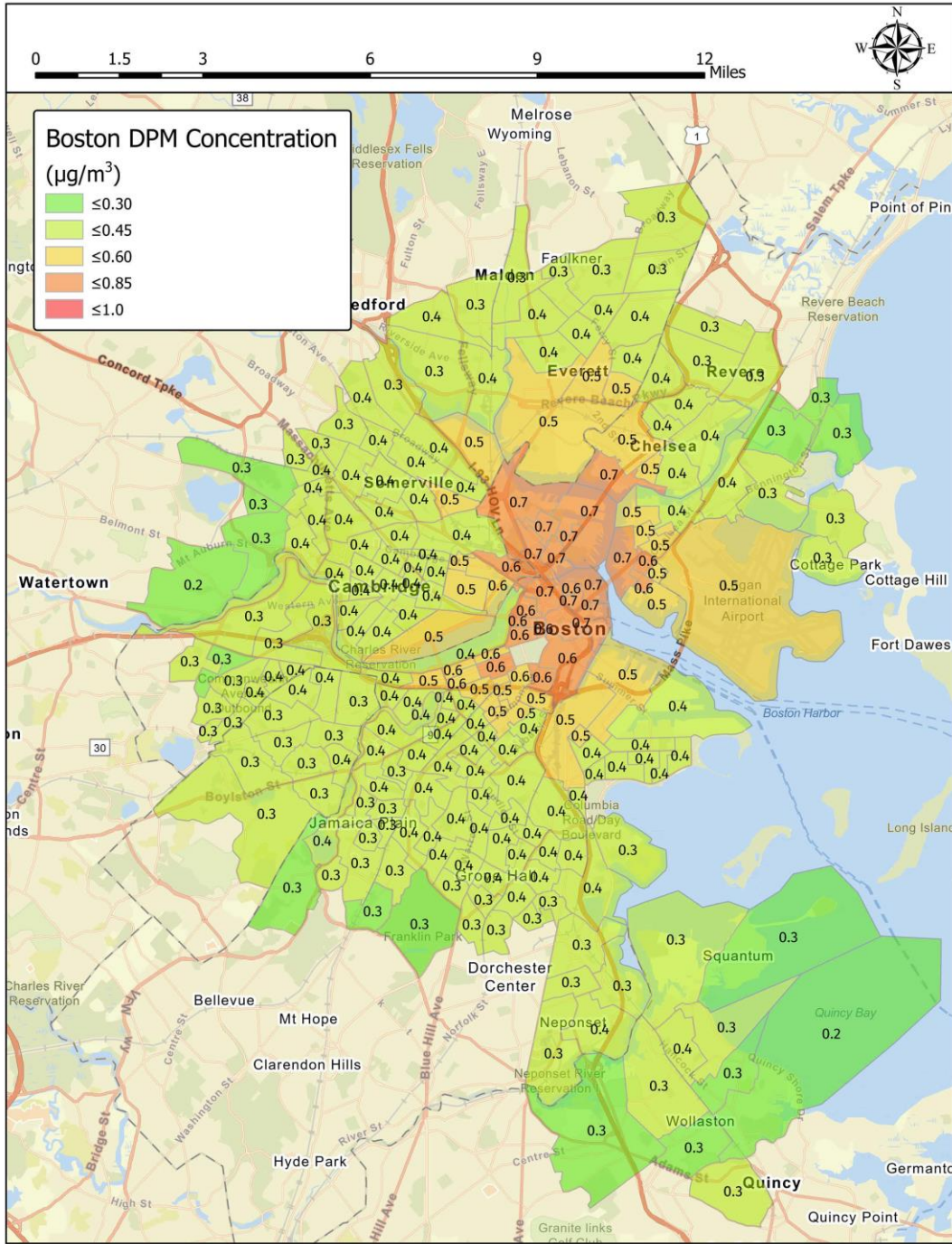
Figure 6-42 shows the baseline DPM concentrations provided by the NATA.

Figure 6-43 shows the baseline DPM-specific cancer risks as determined using the NATA concentration **values and CARB's HARP program**.

Figure 6-44 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Boston community.

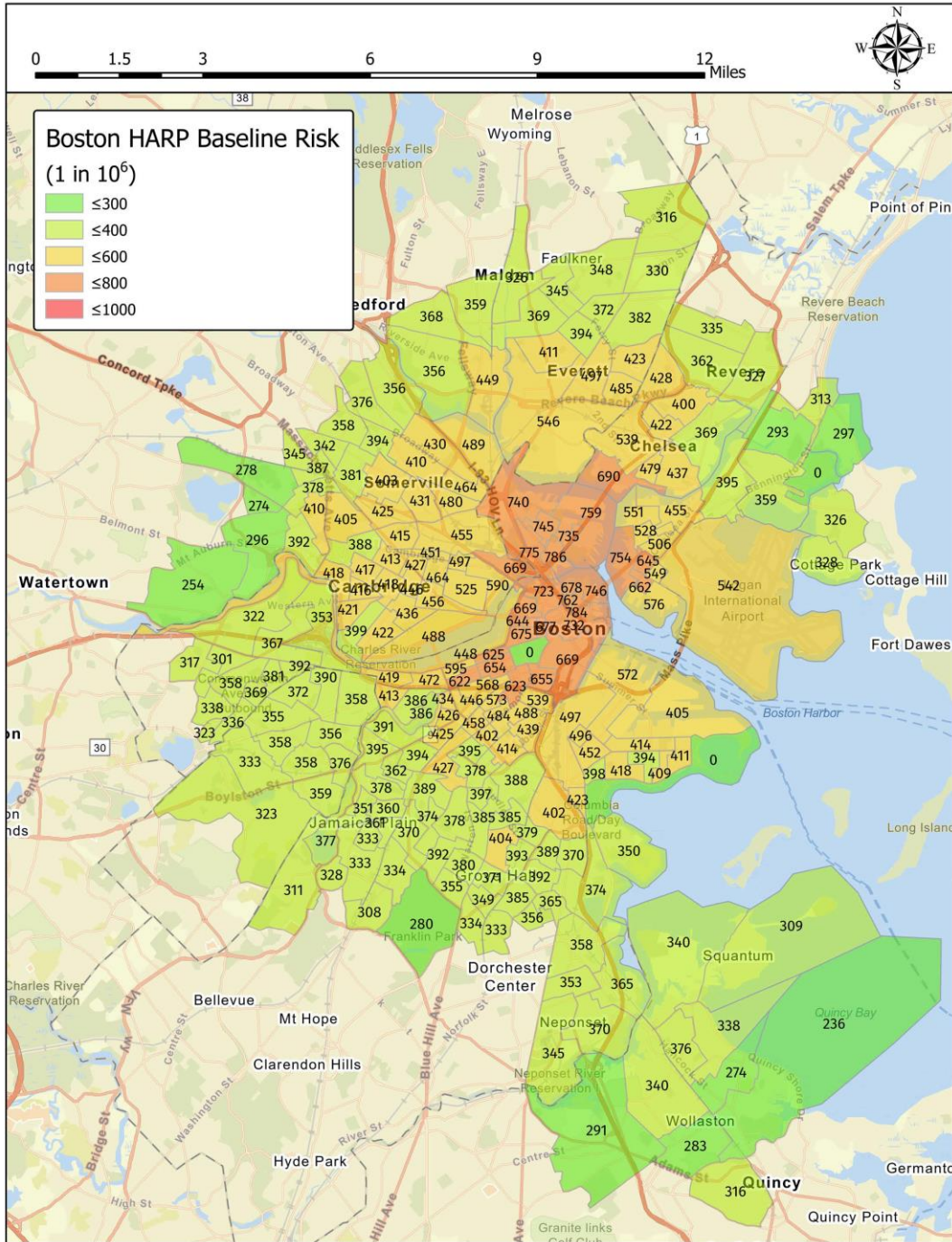
Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the Boston community.

Figure 6-42. Boston Baseline NATA DPM Concentrations



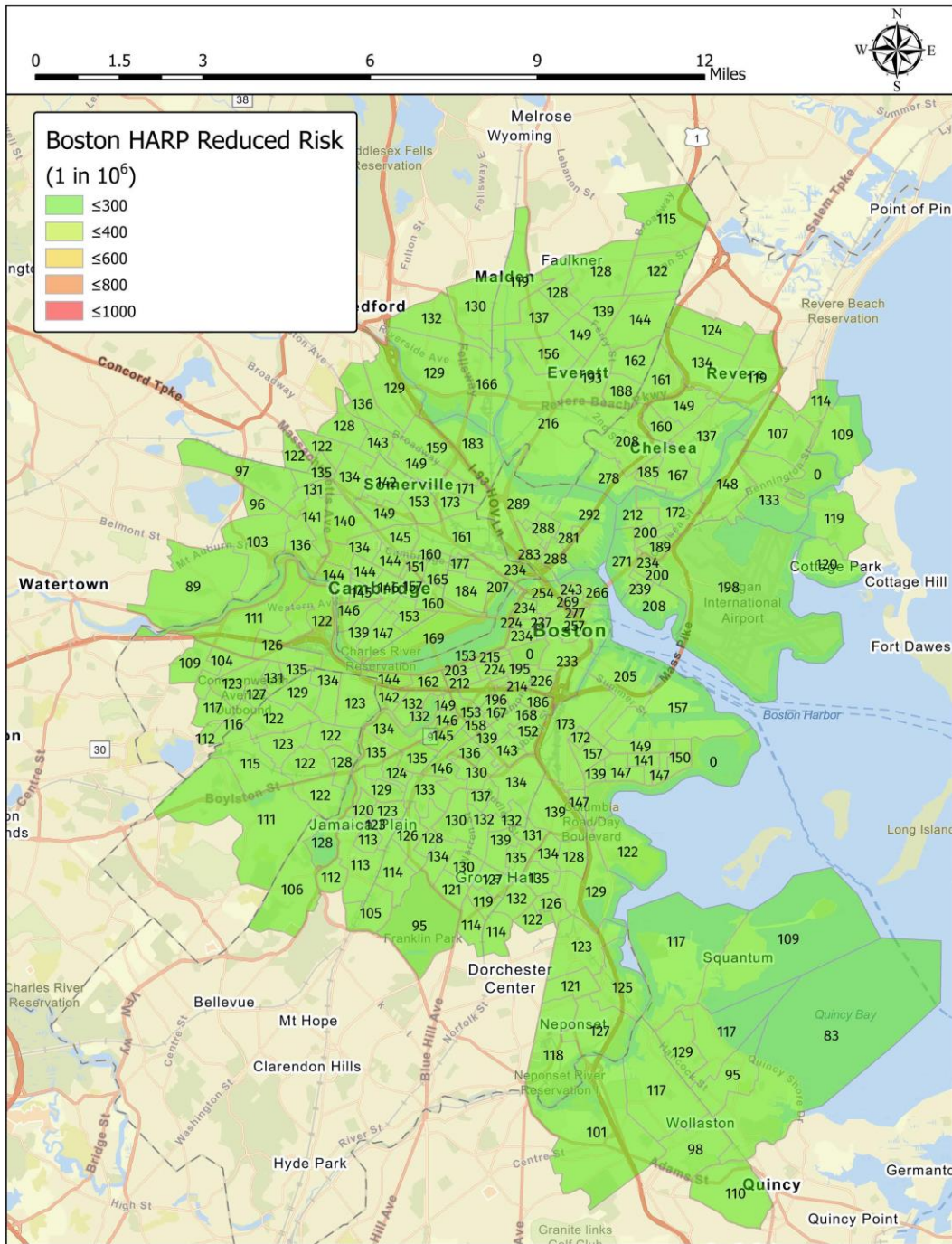
According to the NATA, the maximum baseline DPM concentration in the Boston community is $0.75 \mu\text{g}/\text{m}^3$ for census tracts 25025040100 and 25025030400 with populations of 2,168 and 2,451 residents, respectively. The average DPM concentration of the Boston community is $0.40 \mu\text{g}/\text{m}^3$.

Figure 6-43. Boston Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the Boston community is 786 cancer cases per million residents for census tract 25025040100, with a population of 2,168 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Boston community is 364 cancer cases expected over a 70-year timeline among a total community population of 870,752.

Figure 6-44. Boston Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Boston community becomes 292 cancer cases per million residents for census tract 25025040801, with a population of 3,900 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Boston community becomes 129 cancer case expected over a 70-year timeline among a total community population of 870,752.

6.5.2 Boston Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Boston. The following sources were utilized to generate the HRA.

- Massachusetts State Department of Transportation (MassDOT) – Traffic Counts (2019 Average Annual Daily Traffic)¹⁶

The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-9.

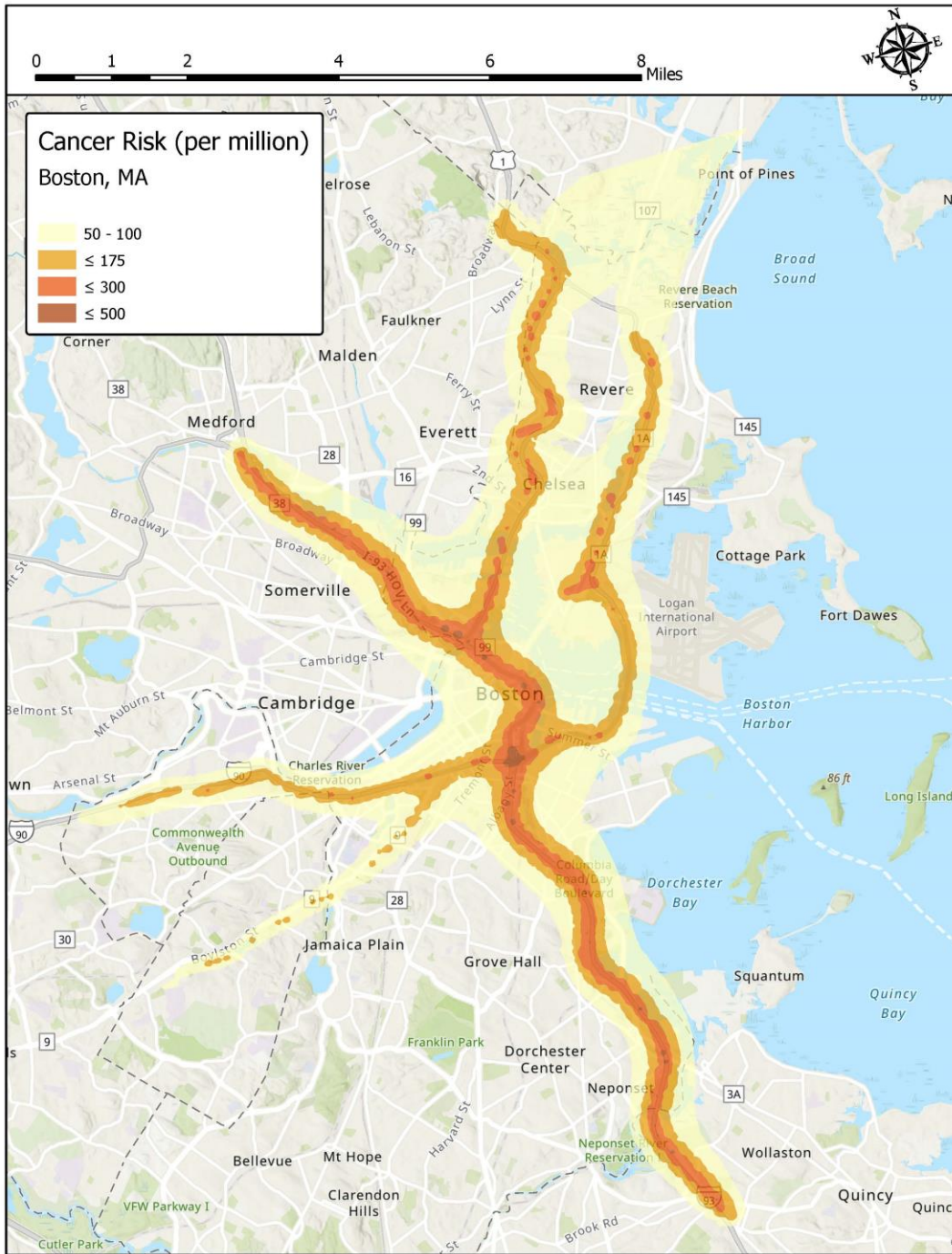
Table 6-9. Boston Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
I-90	I-90 – 58,783 AADT	5,452	59.7%
I-93	I-93 – 124,917 AADT	16,360	59.7%
Route 1	Route 1 – 85,580 AADT	5,763	59.7%
Route 1A	Route 1A – 72,495 AADT	2,787	59.7%
Route 9	Route 9 – 37,285 AADT	1,735	59.7%

These sources were modeled with unit emission rates in AERMOD, and the Table 6-9. listed emission rates **were input into CARB's HARP software to determine cancer risks from the DPM concentrations determined by AERMOD.** While dispersion characteristics remained the same between baseline and reduced modeling scenarios, emission rates were reduced according to the **number of "old technology" engines combusting diesel,** based on source type. The table above shows the **Proportion of "Old Technology" Engine Emissions** where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

¹⁶ <https://mhd.public.ms2soft.com/tcds/tsearch.asp?loc=Mhd&mod=>

Figure 6-45. Boston Baseline Health Risk Assessment Isoleths



The site-specific HRA shows that the point of maximum impact (PMI) is lower than the NATA/HARP evaluation, with an impact of 480 cancer cases per million residents. This PMI does not occur at a residential receptor and does not represent an actual risk to residences in the area. The MEIR occurs at 330,943.6 m E, and 4,691,700.3 m N (NAD 83, UTM Zone 19), with a baseline risk of 379 cancer cases per million residents. This MEIR is also lower than the NATA/HARP hybrid risks evaluated for that census tract (25025030300) with a total risk of 732 in a million. This HRA does not capture all of the cancer-causing

sources in the area but does demonstrate that NATA values are in-line with the site-specific demonstration with some extremely high local maxima due to local residences proximity to highways.

Figure 6-46. Boston Reduced Health Risk Assessment Isopleths



The reduced cancer risk PMI and MEIR are 194 and 153 in one million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 226 in 1 million at the MEIR.

6.5.1 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-10 below.

Table 6-10. Boston Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	54.3	\$1,644,908
Asthma Symptoms - Albuterol use	6,805	\$2,352
ER visits - All Cardiac Outcomes	5.6	\$6,455
ER visits – Respiratory	10.5	\$9,210
HA – All – Respiratory	1.5	\$32,392
HA – Alzheimer’s Disease	20.0	\$254,300
HA – Cardio Cerebro- and Peripheral Vascular Disease	2.8	\$45,079
HA – Parkinson’s Disease	1.7	\$21,701
HA – Respiratory-2	0.5	\$0
HA – Respiratory-2 HA – All Respiratory	2.0	\$0
Incidence – Asthma	53.2	\$2,376,292
Incidence – Hay Fever/Rhinitis	335.6	\$201,353
Incidence – Lung Cancer	3.1	\$38,628
Incidence – Out of Hospital Cardiac Arrest	0.4	\$12,748
Incidence – Stroke	1.2	\$41,997
Minor Restricted Activity Days	19,048	\$1,325,411
Mortality – All Cause	20.5	\$160,819,836
Work Loss Days	3,247	\$794,848
Total		\$167,627,510

6.6 Detroit/Ann Arbor, Michigan

6.6.1 NATA Health Risks

The subsections below review the NATA data available for the Detroit/Ann Arbor, MI (DAA) community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-47, Figure 6-48, and Figure 6-49 show the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

Figure 6-50, Figure 6-51, and Figure 6-52 show those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-53, Figure 6-54, Figure 6-55 show the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the DAA community.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the DAA community.

6.6.1.1 NATA Risk Data

Figure 6-47. North Detroit Baseline NATA Total Cancer Risks

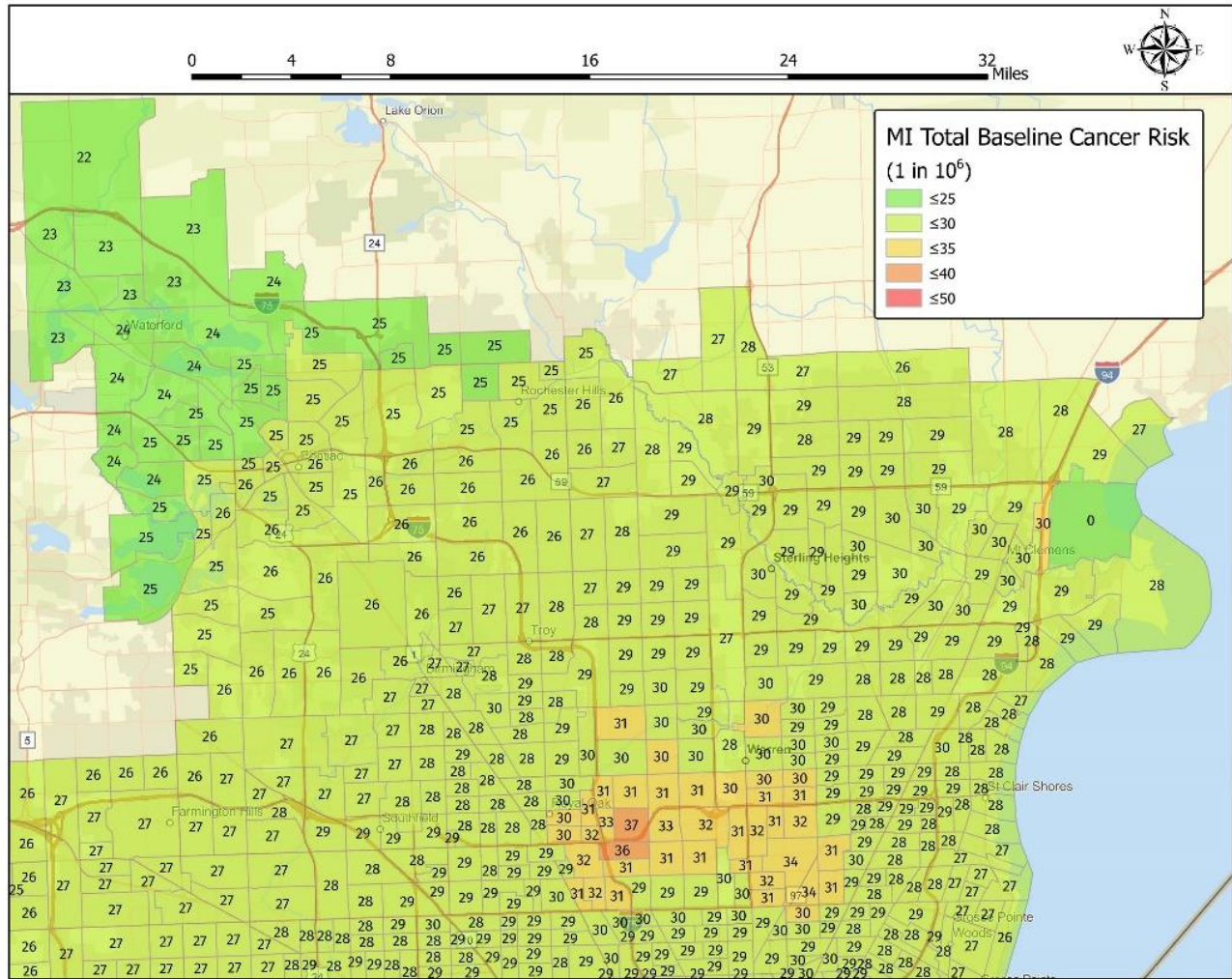


Figure 6-48. South Detroit Baseline NATA Total Cancer Risks

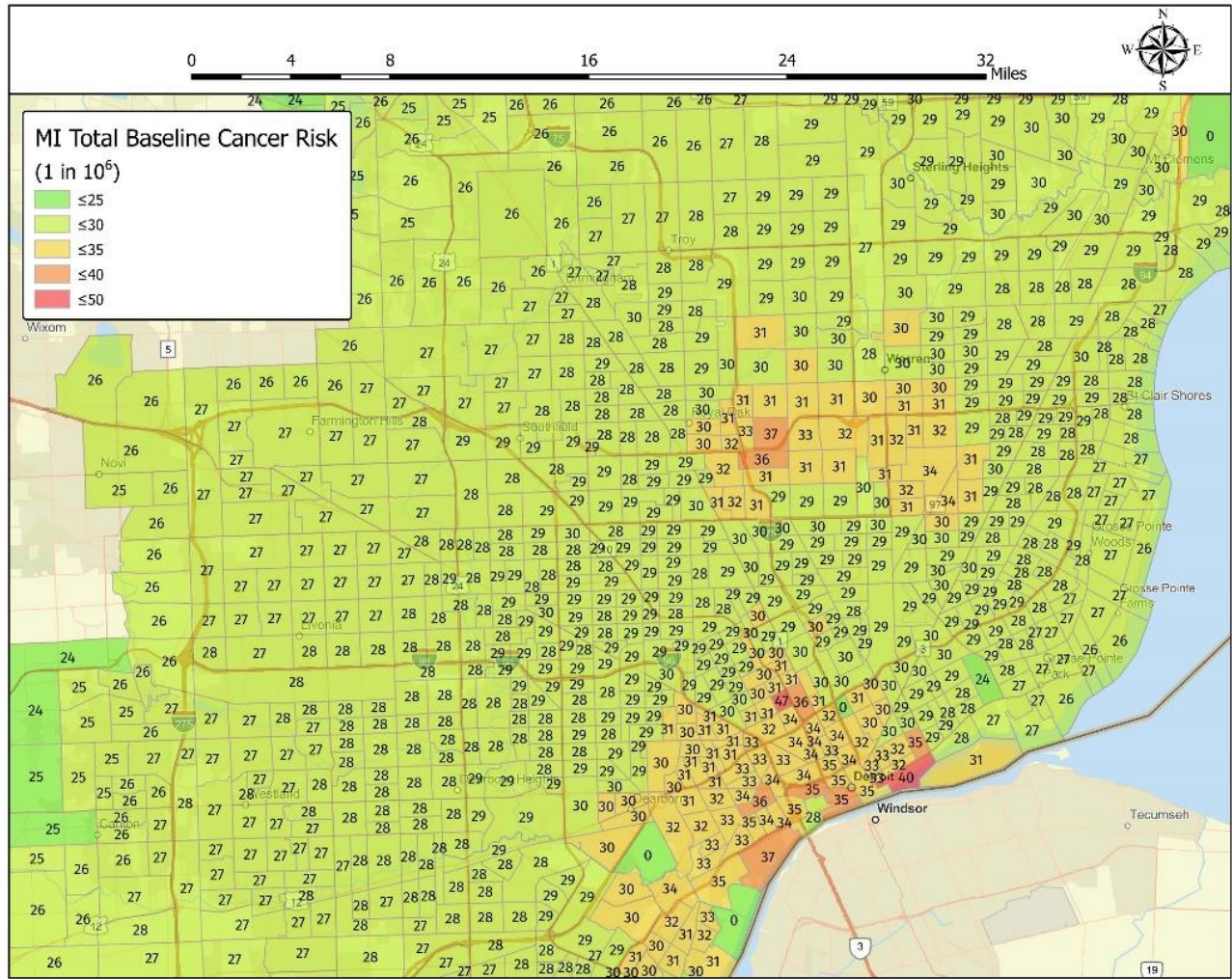
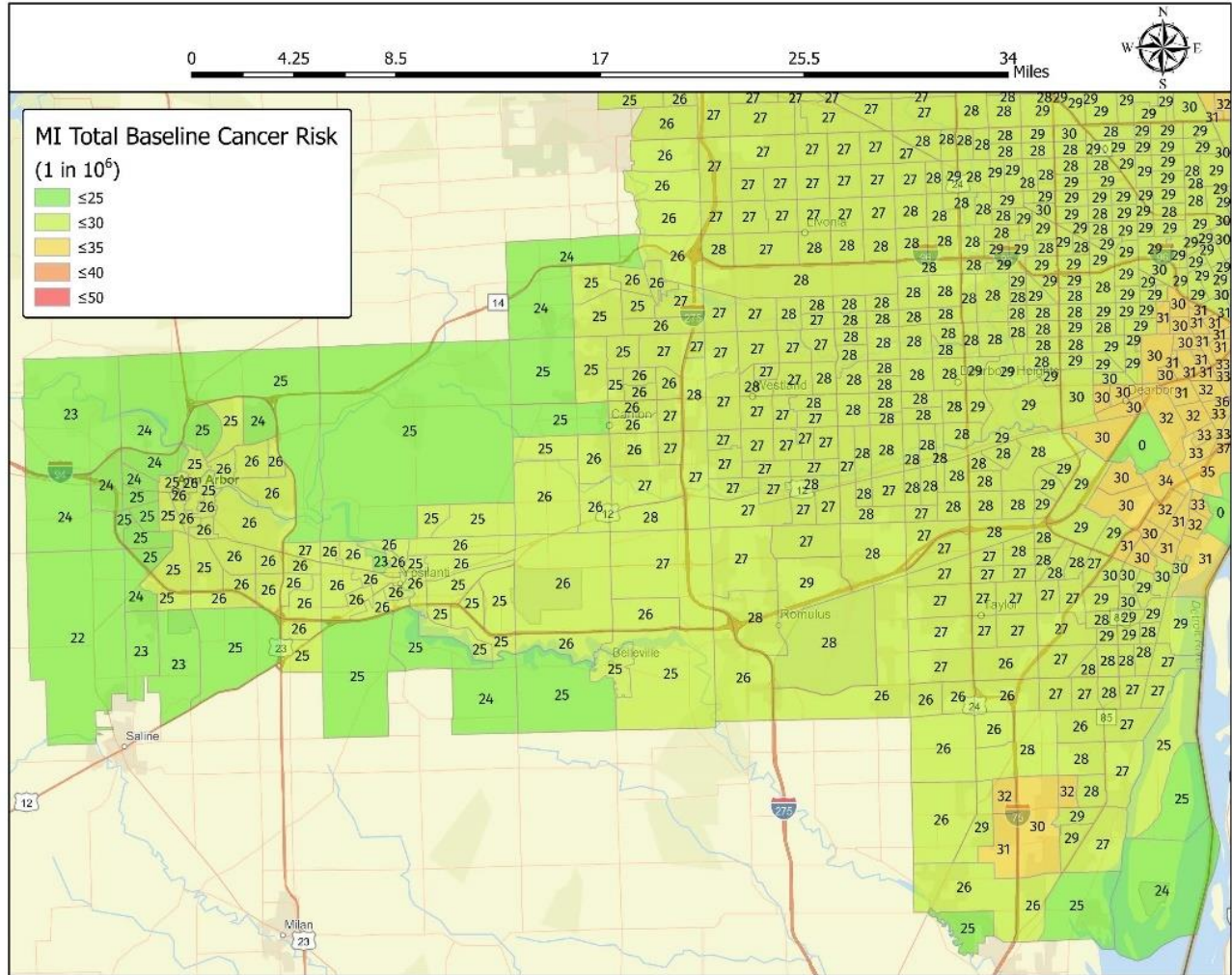


Figure 6-49. Ann Arbor Baseline NATA Total Cancer Risks



According to the NATA, the maximum baseline cancer risk in the DAA community is 46.77 cancer cases per million residents for census tract 26163532600, with a population of 2,043 residents. When accounting for all of the communities assessed, the total cancer burden for the DAA community is 98 cancer cases expected over a 70-year timeline among a total community population of 3,502,408.

Figure 6-50. North Detroit Baseline NATA DPM Cancer Risks

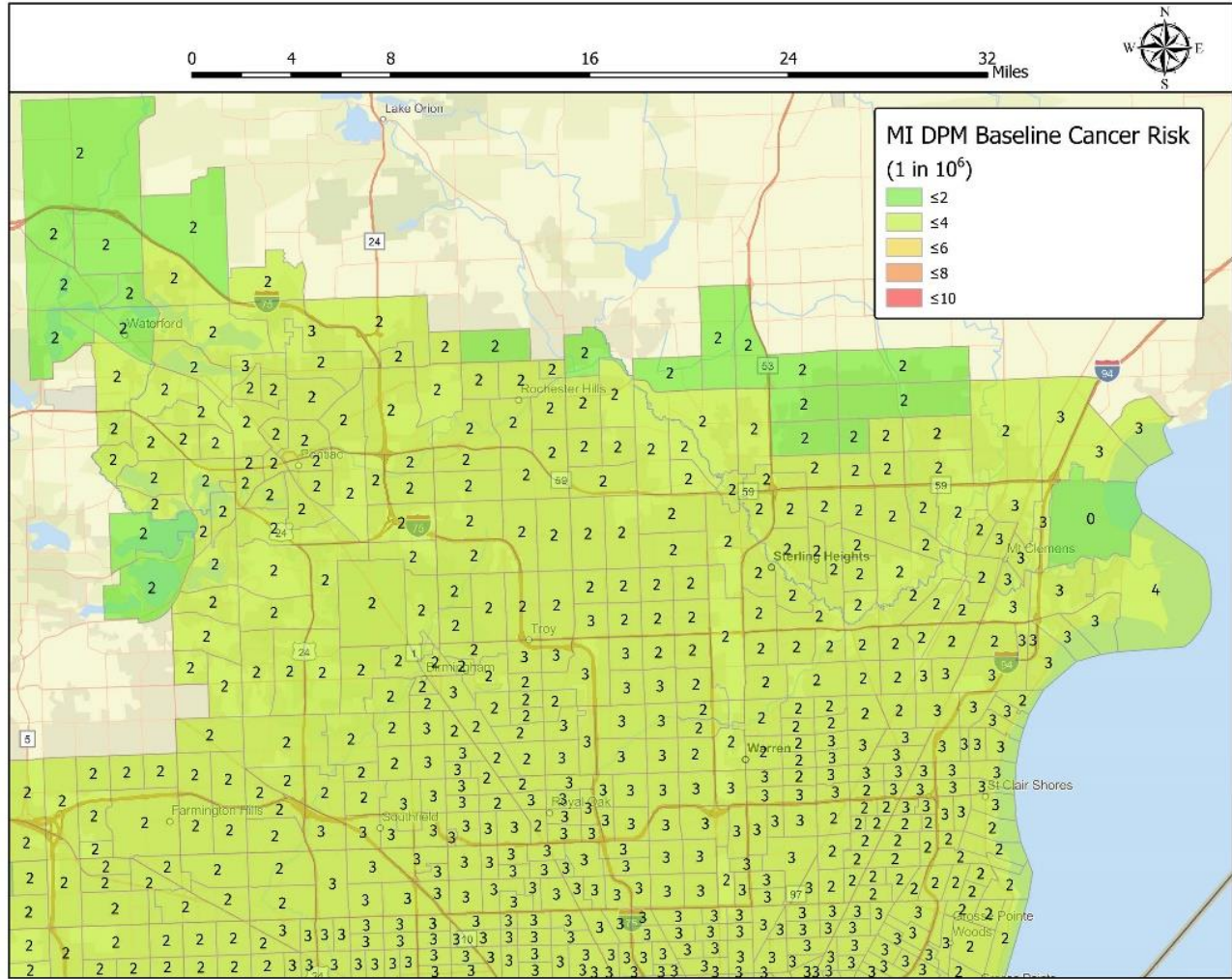


Figure 6-51. South Detroit Baseline NATA DPM Cancer Risks

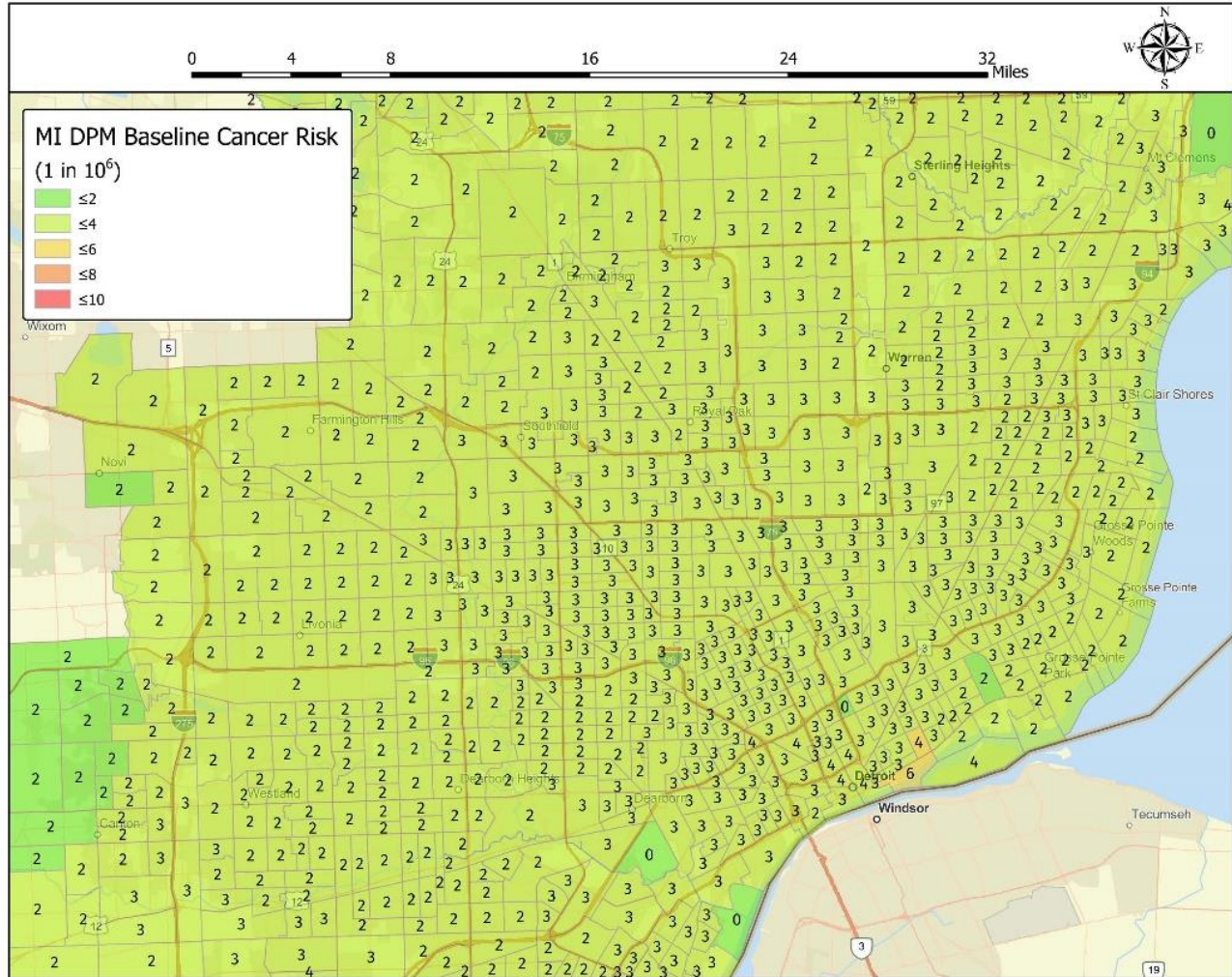
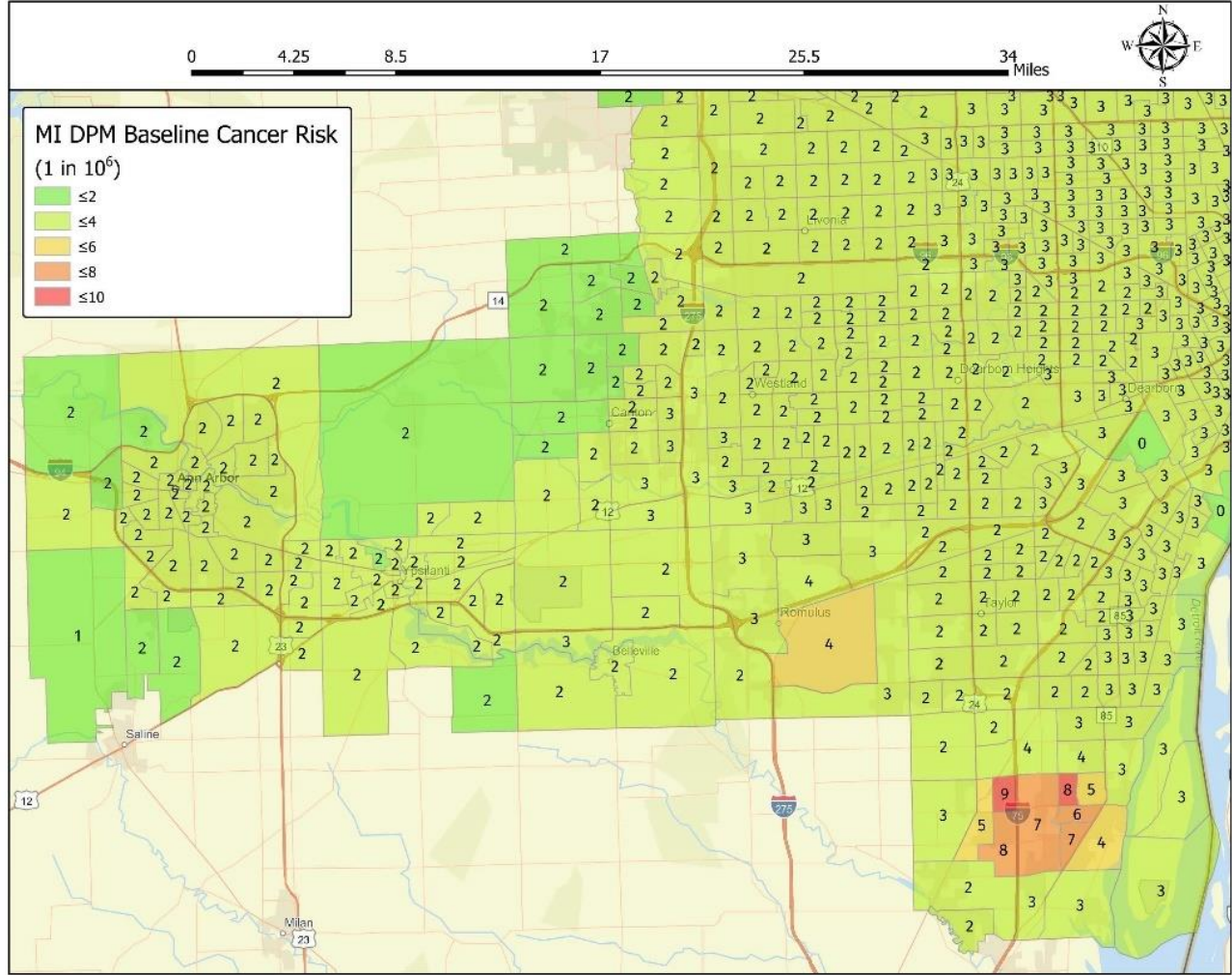


Figure 6-52. Ann Arbor Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the DAA community is 9 cancer cases per million residents for census tract 26163593200, with a population of 3,934 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the DAA community is 9 cancer cases expected over a 70-year timeline among a total community population of 3,502,408.

Figure 6-53. North Detroit Reduced NATA DPM Cancer Risks

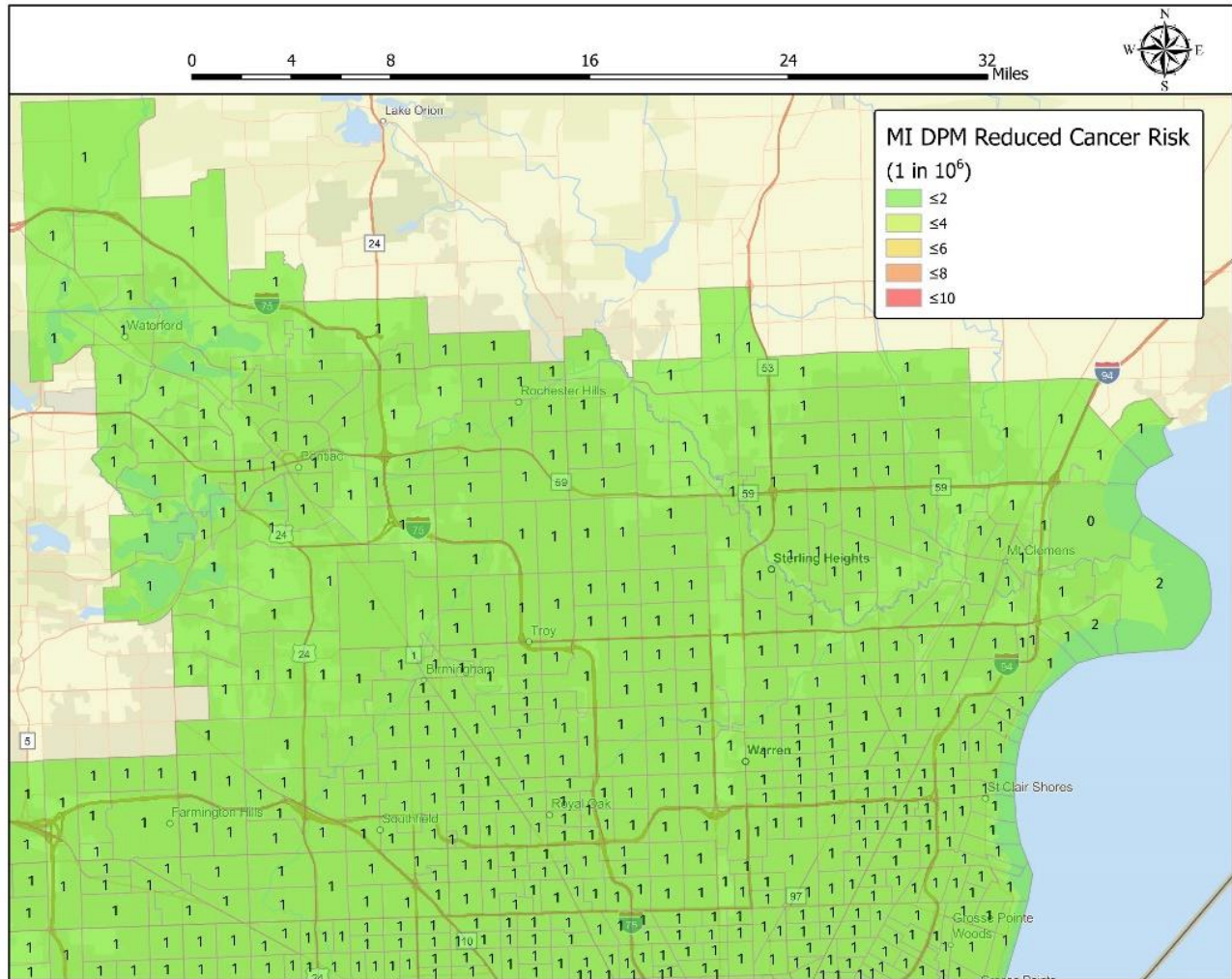


Figure 6-54. South Detroit Reduced NATA DPM Cancer Risks

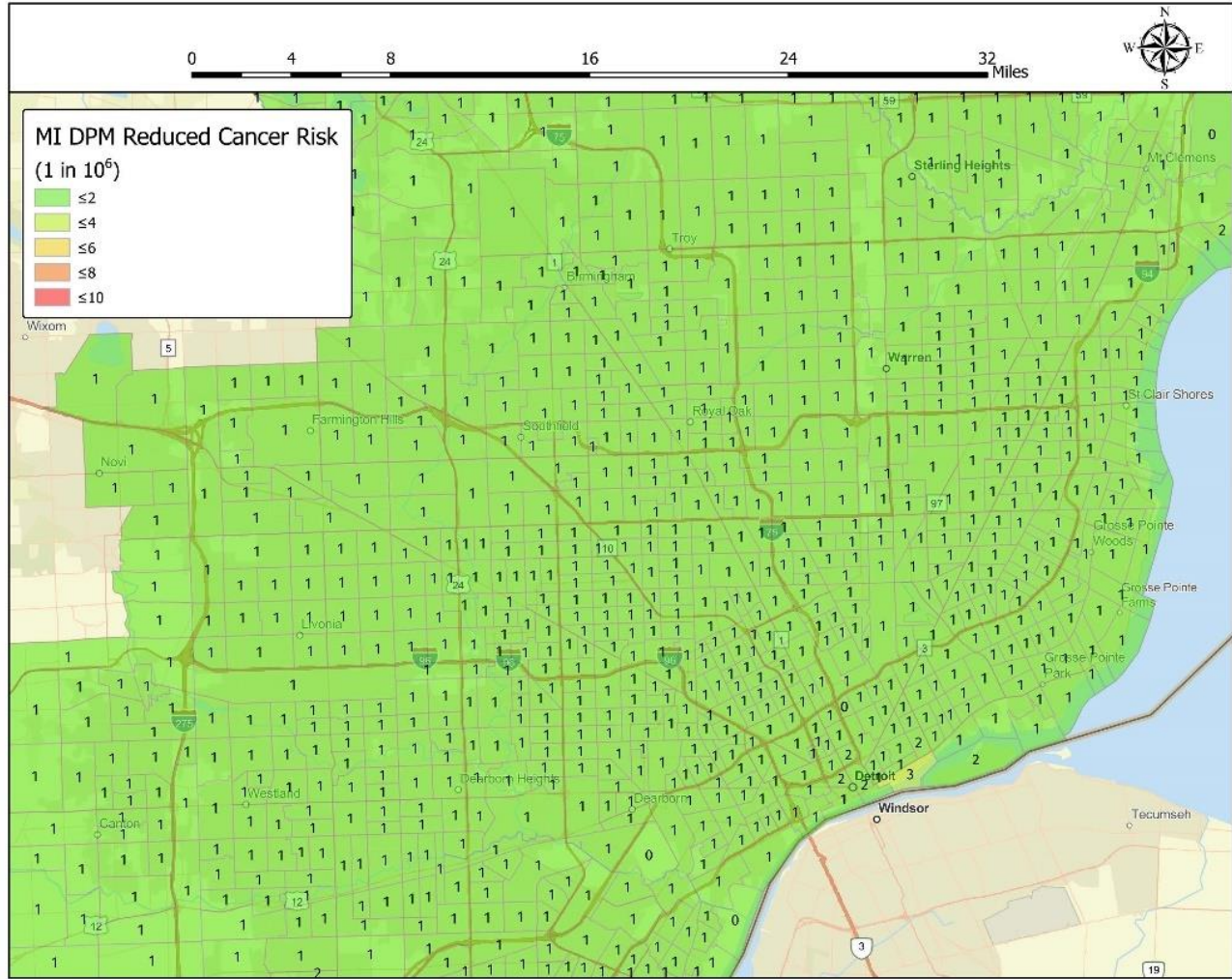
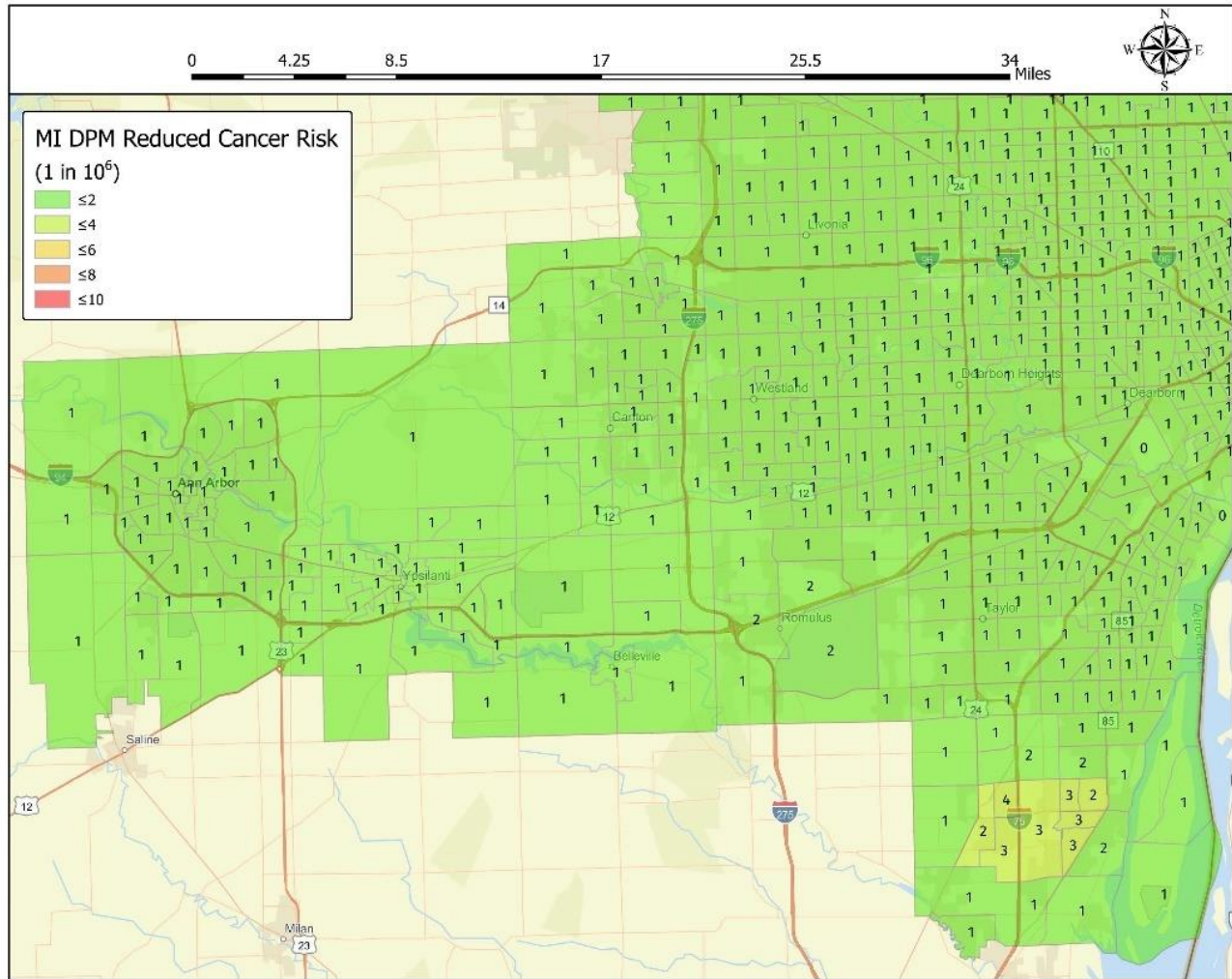


Figure 6-55. Ann Arbor Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the DAA community becomes 4 cancer cases per million residents for census tract 26163593200, with a population of 3,934 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the DAA community becomes 4 cancer cases expected over a 70-year timeline among a total community population of 3,502,408.

6.6.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB’s HARP program with OEHHA cancer unit risk values.** The data is outlined in the following order:

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program.**

Figure 6-56, Figure 6-57, Figure 6-59, and Figure 6-58 show the baseline DPM concentrations provided by the NATA.

Figure 6-59, Figure 6-60, and Figure 6-61 show the baseline DPM-specific cancer risks as determined using **the NATA concentration values and CARB's HARP program.**

Figure 6-62, Figure 6-63, and Figure 6-64 show the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the DAA community.

Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the DAA community.

Figure 6-56. North Detroit Baseline NATA DPM Concentrations

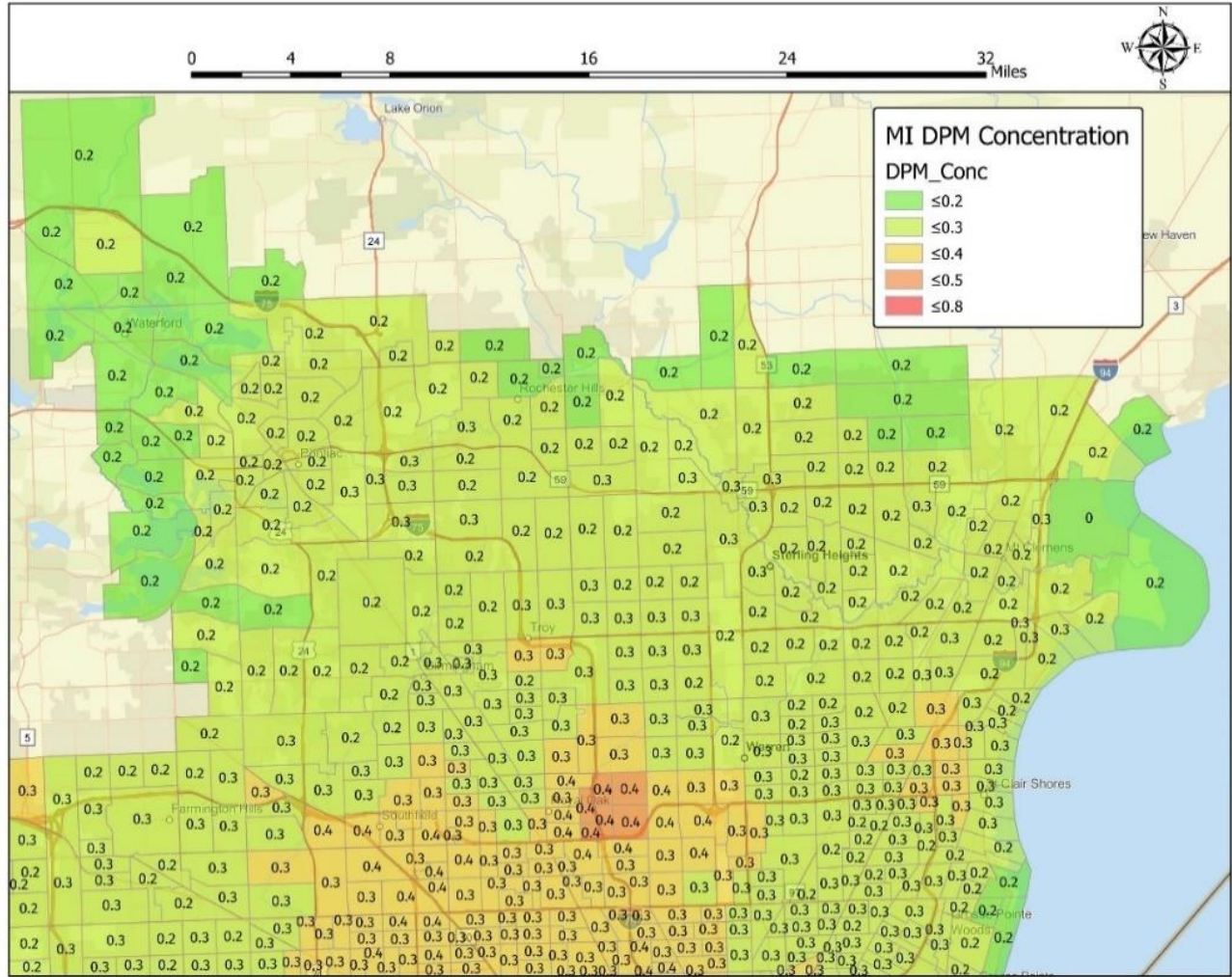


Figure 6-57. South Detroit Baseline NATA DPM Concentrations

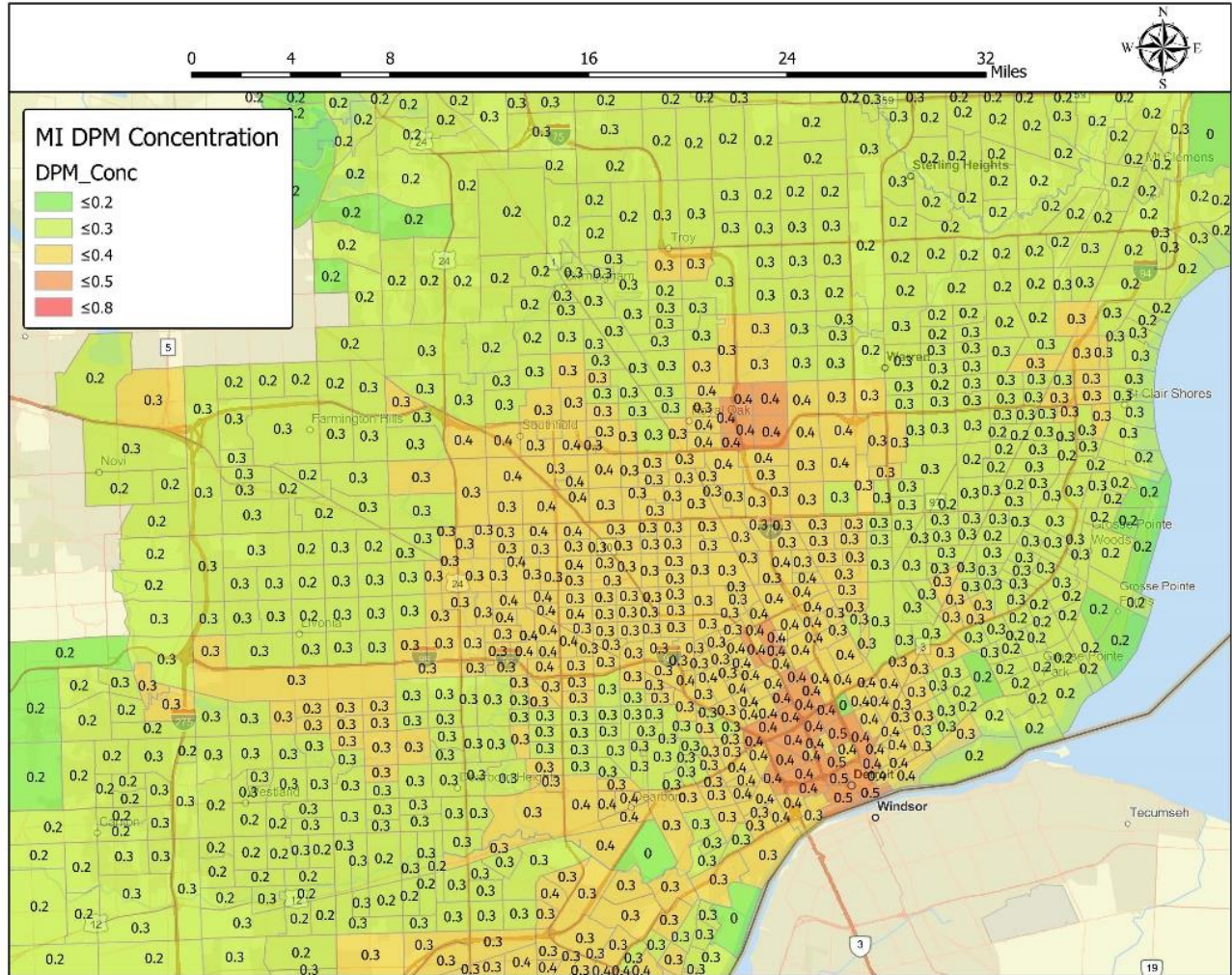
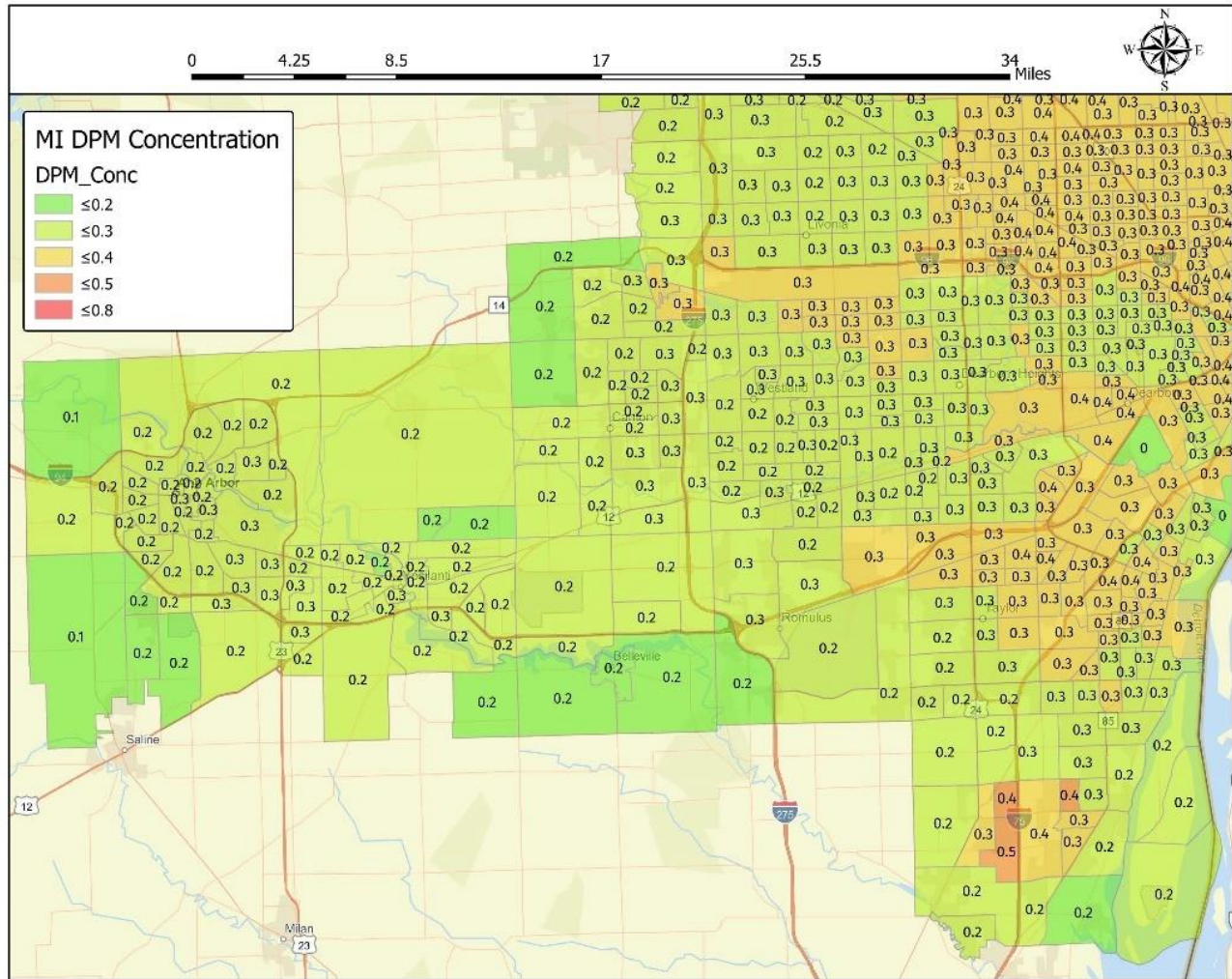


Figure 6-58. Ann Arbor Baseline NATA DPM Concentrations



According to the NATA, the maximum baseline DPM concentration in the DAA community is $0.47 \mu\text{g}/\text{m}^3$ for census tract 26163520800, with a population of 1,616 residents. The average DPM concentration of the Detroit community is $0.28 \mu\text{g}/\text{m}^3$.

Figure 6-59. North Detroit Baseline NATA/HARP DPM Hybrid Risks

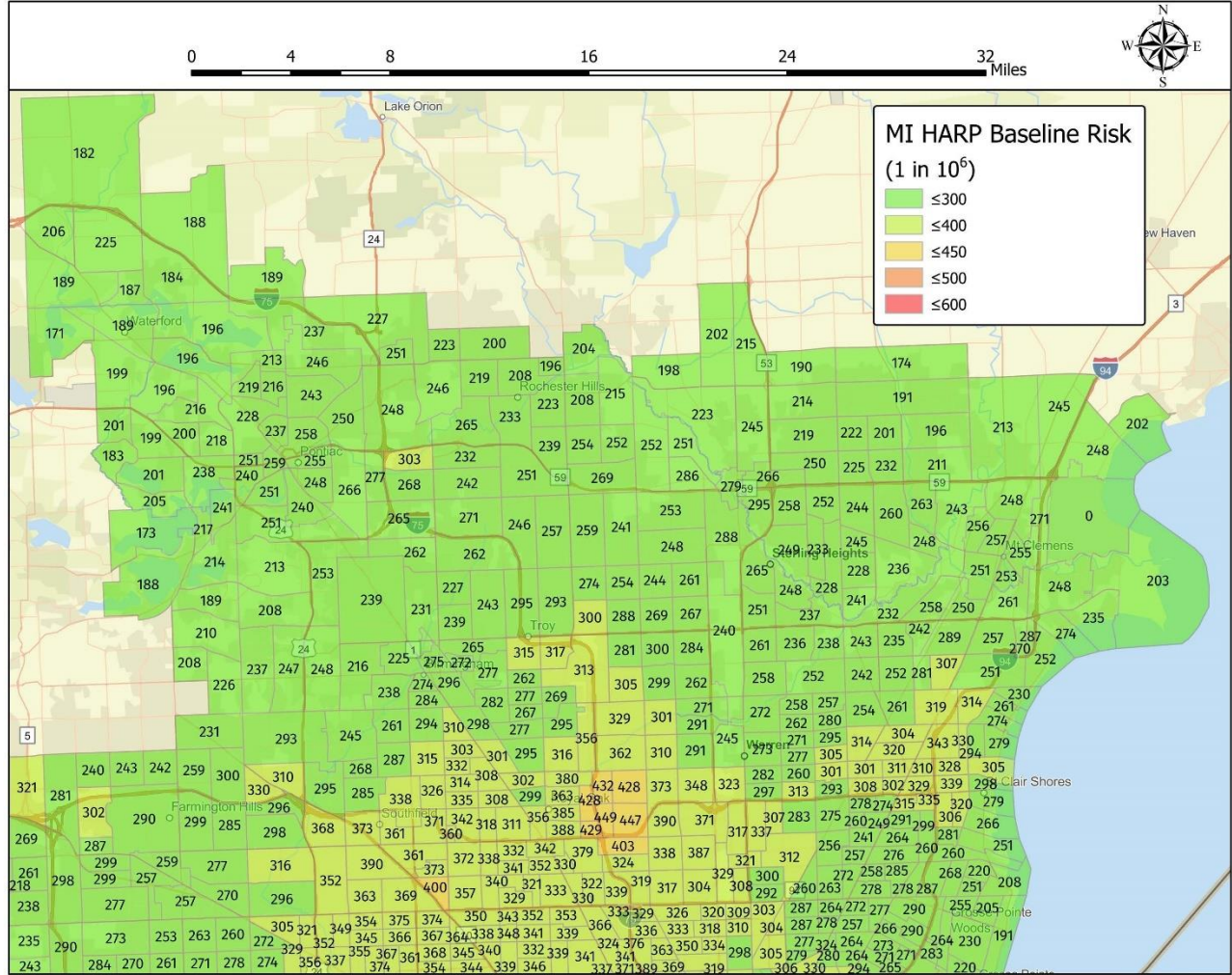


Figure 6-60. South Detroit Baseline NATA/HARP DPM Hybrid Risks

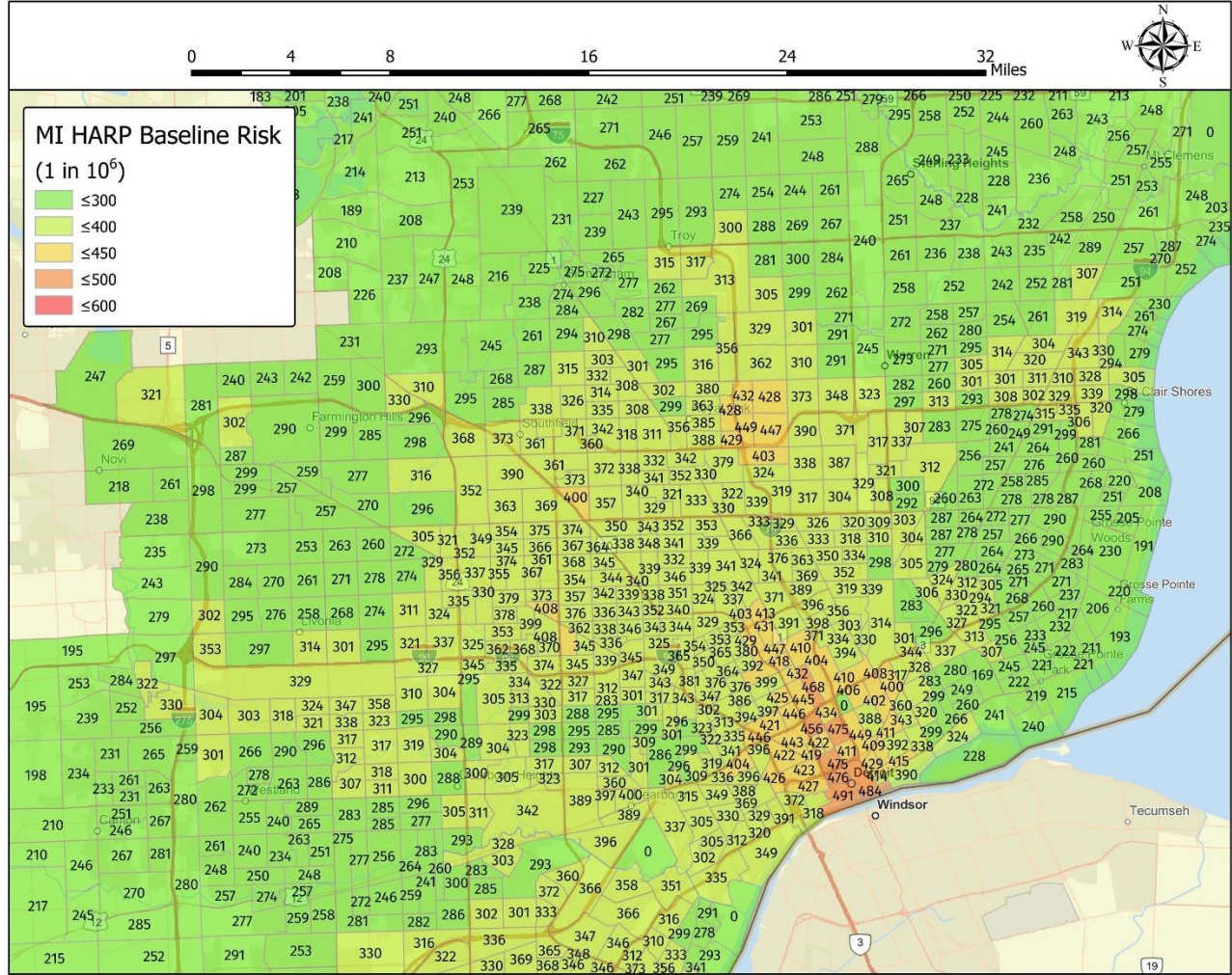
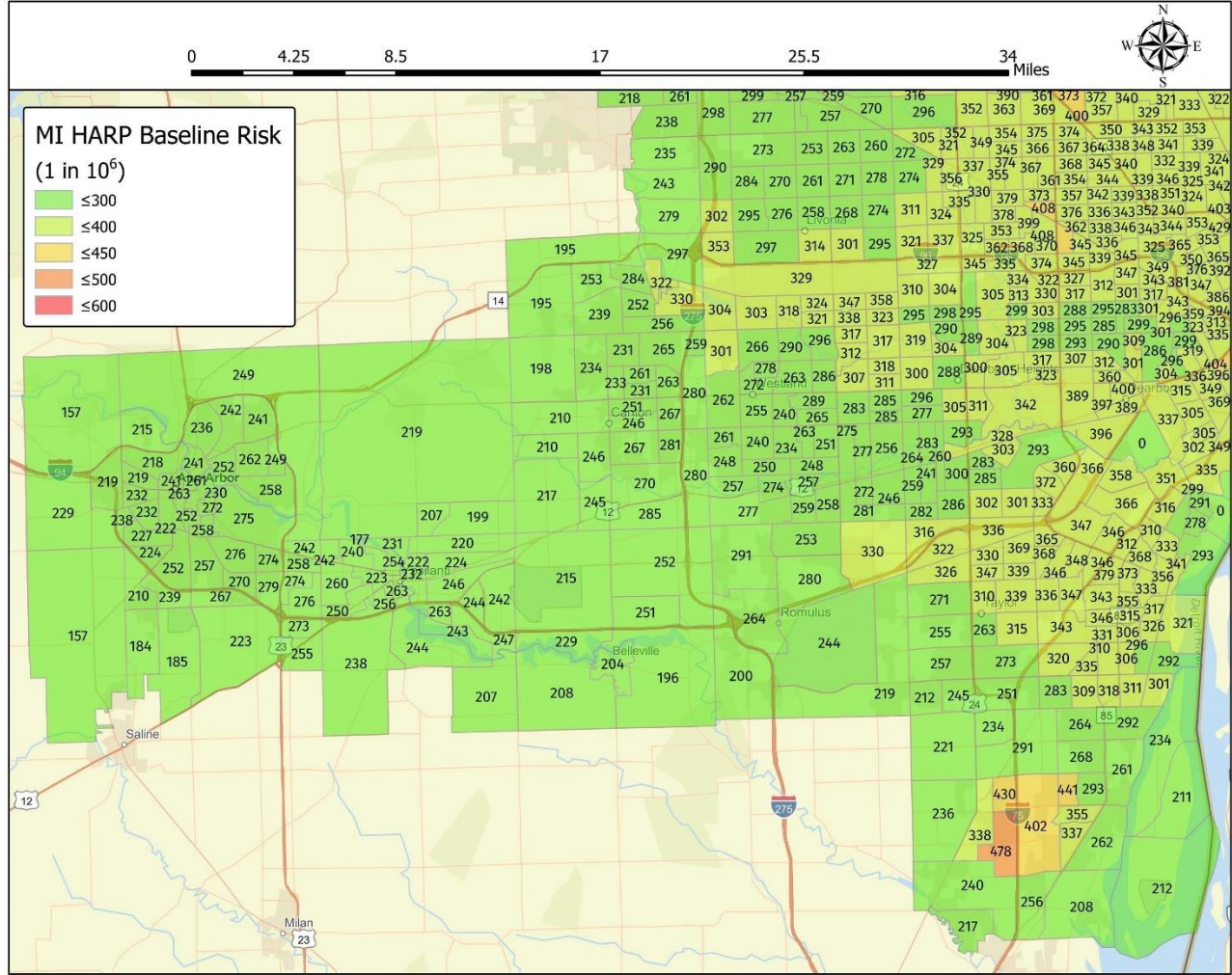


Figure 6-61. Ann Arbor Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the DAA community is 491 cancer cases per million residents for census tract 26163520800, with a population of 1,616 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Detroit community is 1,007 cancer cases expected over a 70-year timeline among a total community population of 3,502,408.

Figure 6-62. North Detroit Reduced NATA/HARP DPM Hybrid Risks

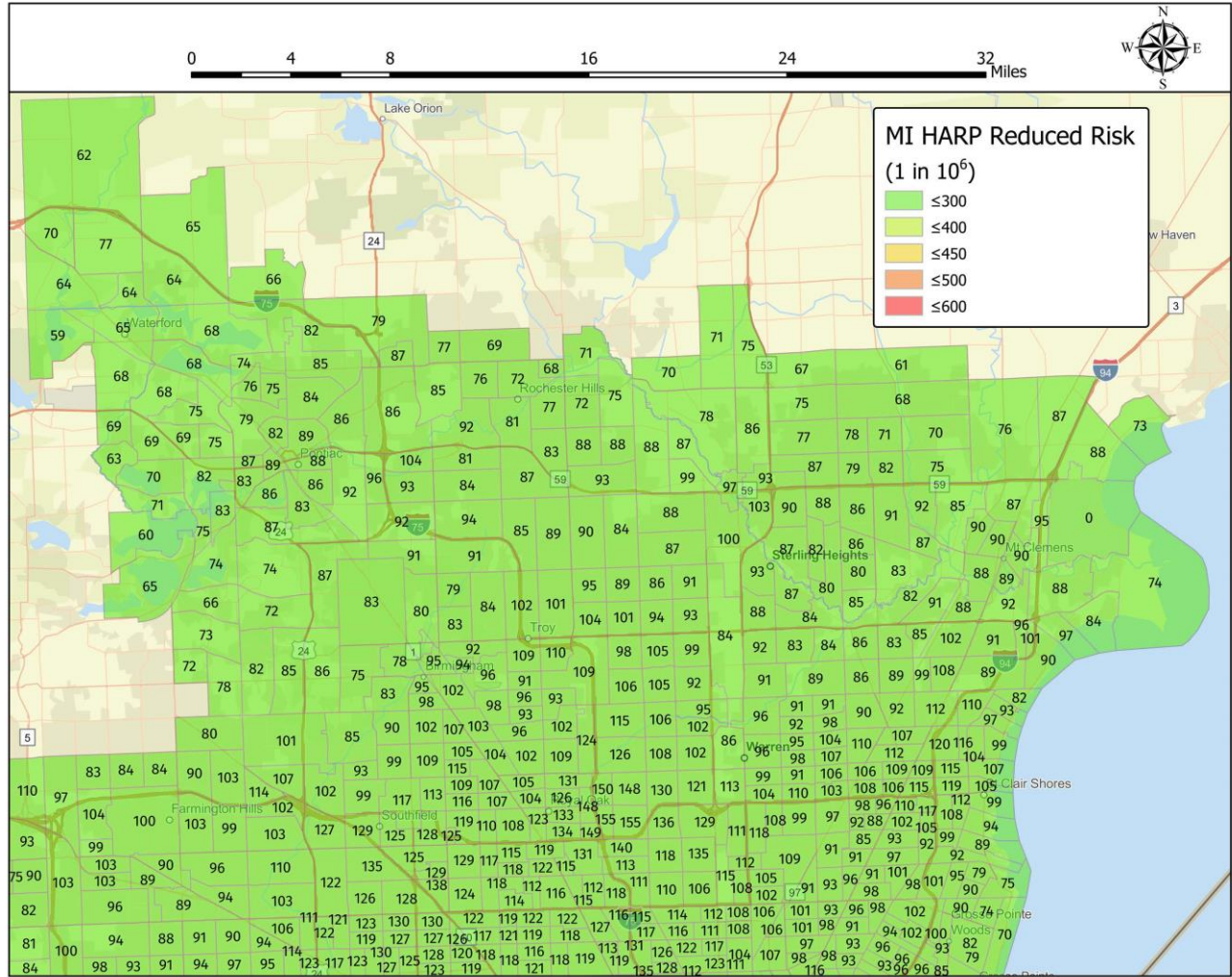


Figure 6-63. South Detroit Reduced NATA/HARP DPM Hybrid Risks

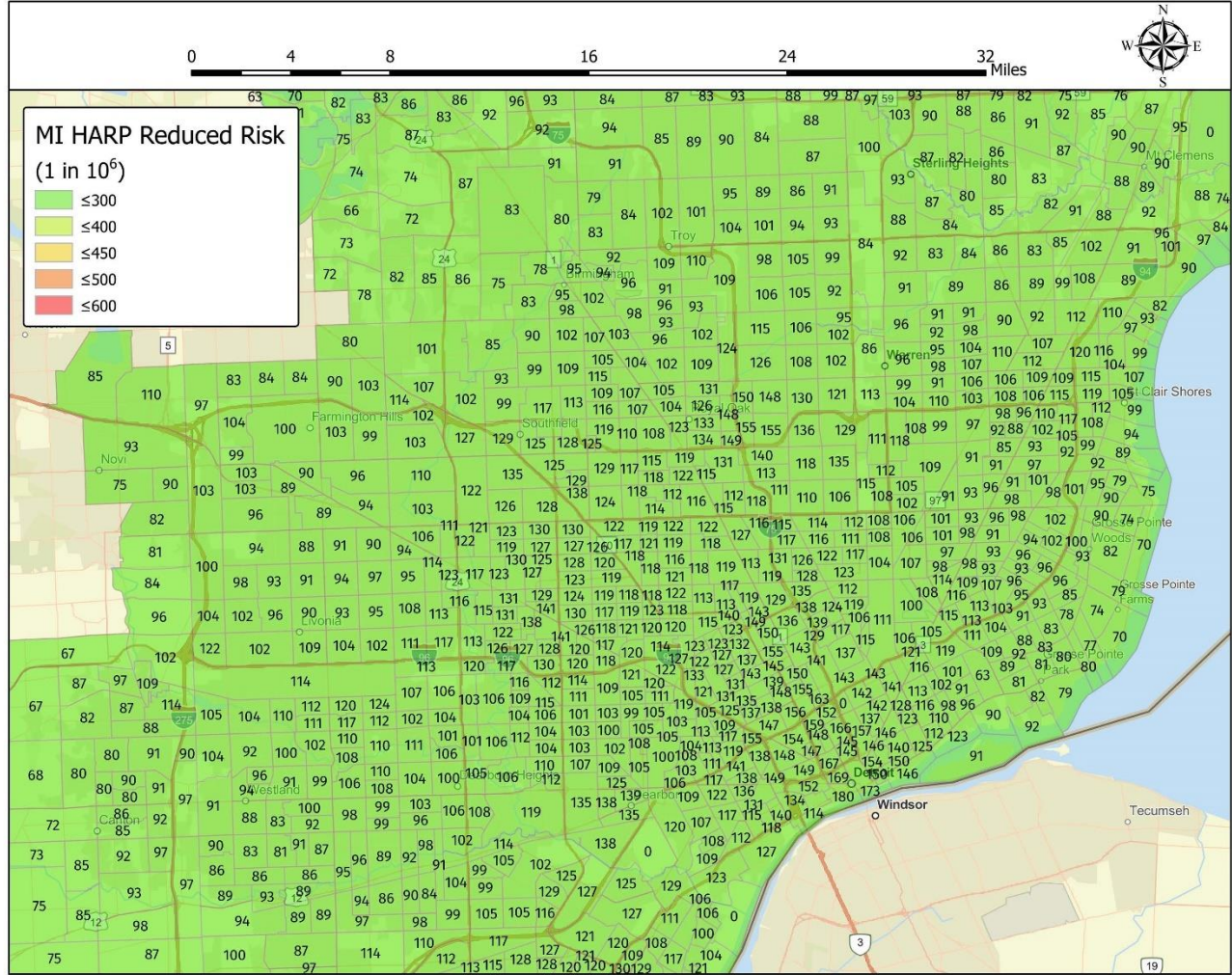
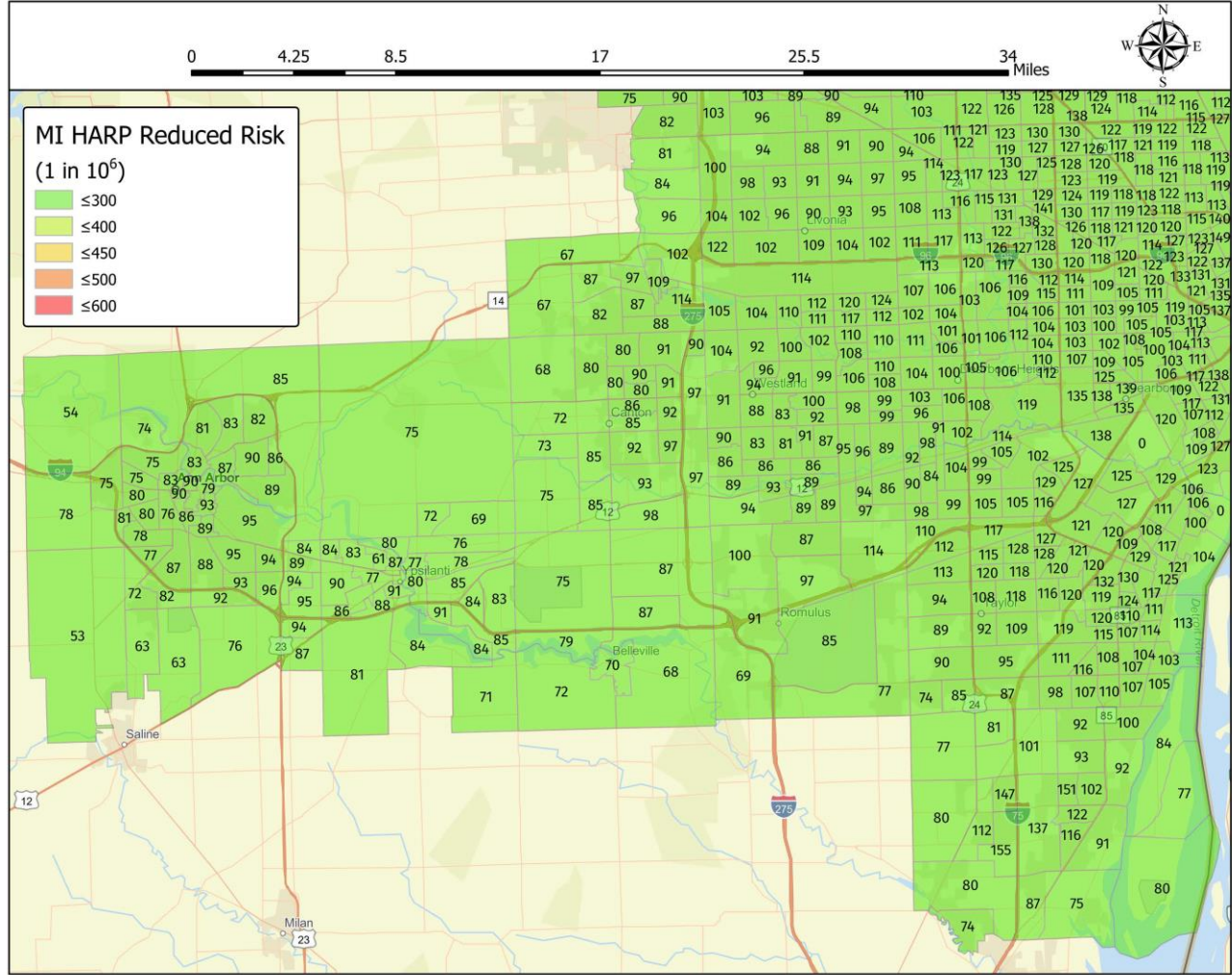


Figure 6-64. Ann Arbor Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Detroit community becomes 180 cancer cases per million residents for census tract 26163520800, with a population of 1,616 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Detroit community becomes 351 cancer case expected over a 70-year timeline among a total community population of 3,502,408.

6.6.2 Detroit Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Detroit. The following sources were utilized to generate the HRA.

- Michigan Department of Transportation (MDOT) – Traffic Counts (2019 Average Annual Daily Traffic)¹⁷

The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-11.

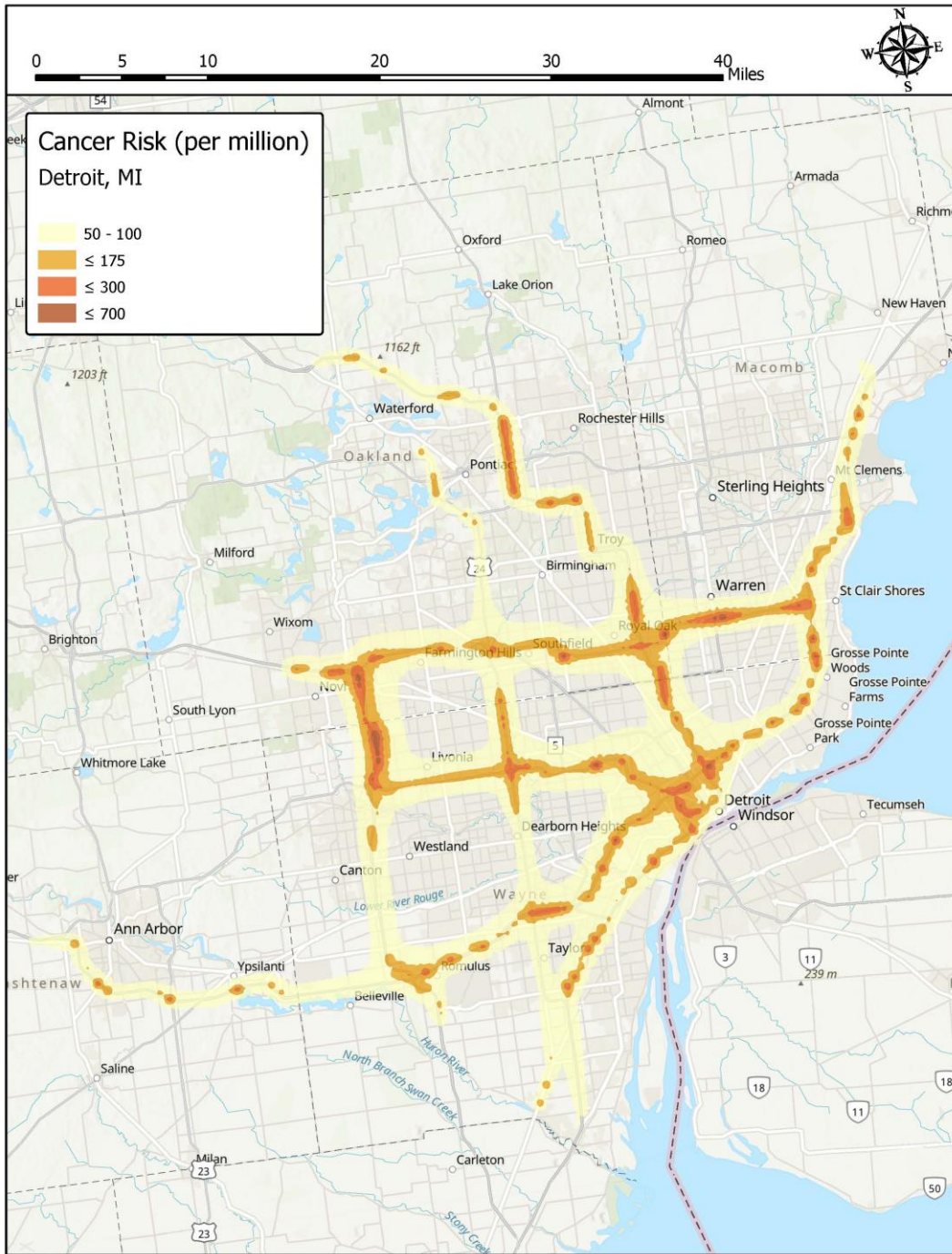
Table 6-11. Detroit Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
I-75	I-75 – 88,385 AADT	58,099	59.7%
I-94	I-94 – 95,922 AADT	71,176	59.7%
I-275	I-275 – 97,964 AADT	22,223	59.7%
I-696	I-696 – 115,640 AADT	32,680	59.7%
I-96	I-96 – 121,230 AADT	38,034	59.7%
M24	M-24 – 45,043 AADT	18,641	59.7%

These sources were modeled with unit emission rates in AERMOD, and the Table 6-11 Table 6-11. listed emission rates **were input into CARB's HARP software to determine cancer** risks from the DPM concentrations determined by AERMOD. While dispersion characteristics remained the same between **baseline and reduced modeling scenarios, emission rates were reduced according to the number of "old technology" engines combusting diesel**, based on source type. The table above shows the **Proportion of "Old Technology" Engine Emissions** where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

¹⁷ <https://lrs.state.mi.us/portal/apps/webappviewer/index.html?id=1a8bf6b2681d483ca9090ebec5d105ff>

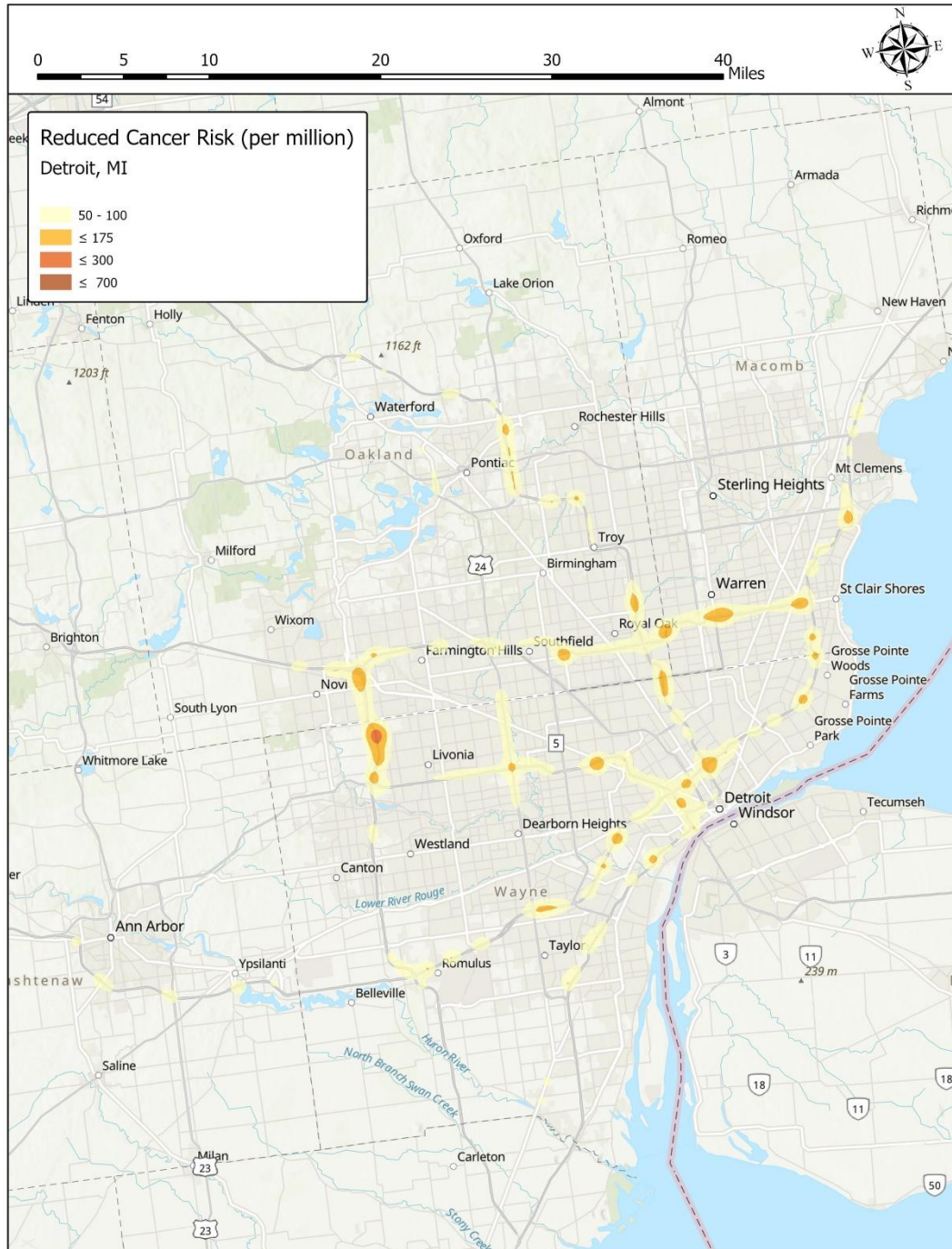
Figure 6-65. Detroit/Ann Arbor Baseline Health Risk Assessment Isoleths



The site-specific HRA shows that the point of maximum impact (PMI) is substantially higher than the NATA/HARP evaluation, with an impact of 684 cancer cases per million residents. This PMI does not occur at a residential receptor, though, and does not represent an actual risk to residences in the area. The MEIR occurs at 334,174.4 m E, and 4,706,498.6 m N (NAD 83, UTM Zone 17), with a baseline risk of 470 cancer cases per million residents. This MEIR is higher than the NATA/HARP hybrid risks evaluated for that census tract (41051001900) with a total risk of 297 in a million. This HRA does not capture all of the cancer-causing

sources in the area but does demonstrate that NATA values are in-line with the site-specific demonstration with some extremely high local maxima due to local residences proximity to highways.

Figure 6-66. Detroit/Ann Arbor Reduced Health Risk Assessment Isopeleths



The reduced cancer risk PMI and MEIR are 276 and 189 in 1 million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 281 in 1 million at the MEIR.

6.6.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-12 below.

Table 6-12. Detroit Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	107.6	\$3,373,685
Asthma Symptoms - Albuterol use	9,725	\$3,361
ER visits - All Cardiac Outcomes	9.6	\$11,115
ER visits – Respiratory	19.6	\$17,099
HA – All – Respiratory	2.4	\$50,024
HA – Alzheimer’s Disease	14.3	\$174,578
HA – Cardio Cerebro- and Peripheral Vascular Disease	4.9	\$75,614
HA – Parkinson’s Disease	2.0	\$25,402
HA – Respiratory-2	0.7	\$0
HA – Respiratory-2 HA – All Respiratory	3.2	\$0
Incidence – Asthma	72.3	\$3,228,747
Incidence – Hay Fever/Rhinitis	467.0	\$280,130
Incidence – Lung Cancer	4.1	\$51,653
Incidence – Out of Hospital Cardiac Arrest	0.5	\$16,725
Incidence – Stroke	1.7	\$57,566
Minor Restricted Activity Days	22,468	\$1,563,382
Mortality – All Cause	33.0	\$257,662,511
Work Loss Days	3,781	\$697,926
Total		\$267,289,517

6.7 Minneapolis/St. Paul, Minnesota

6.7.1 NATA Health Risks

The subsections below review the NATA data available for the Minneapolis/St. Paul, MN (MSP) community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-67 shows the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

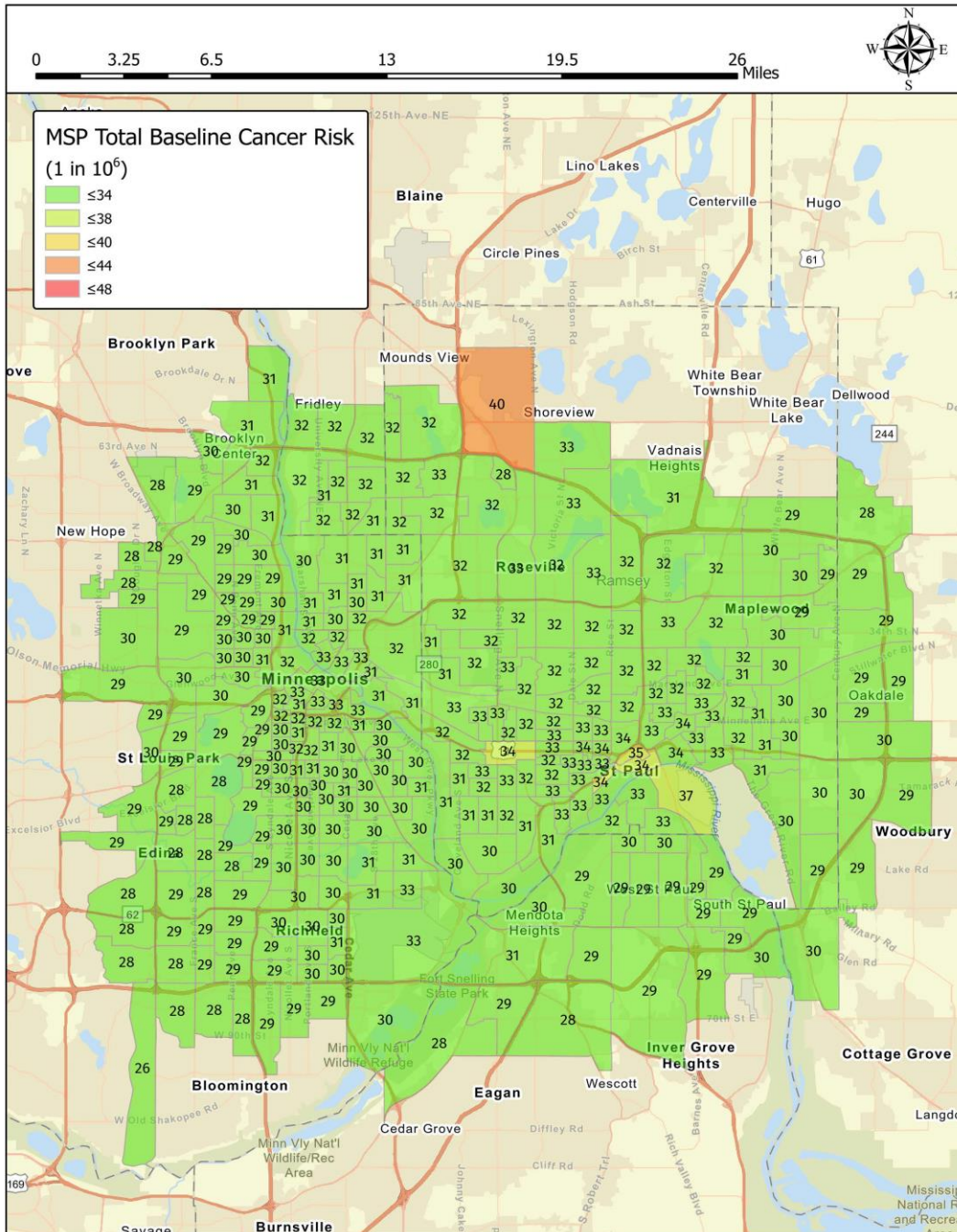
Figure 6-68 shows those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-69 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the MSP community.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the MSP community.

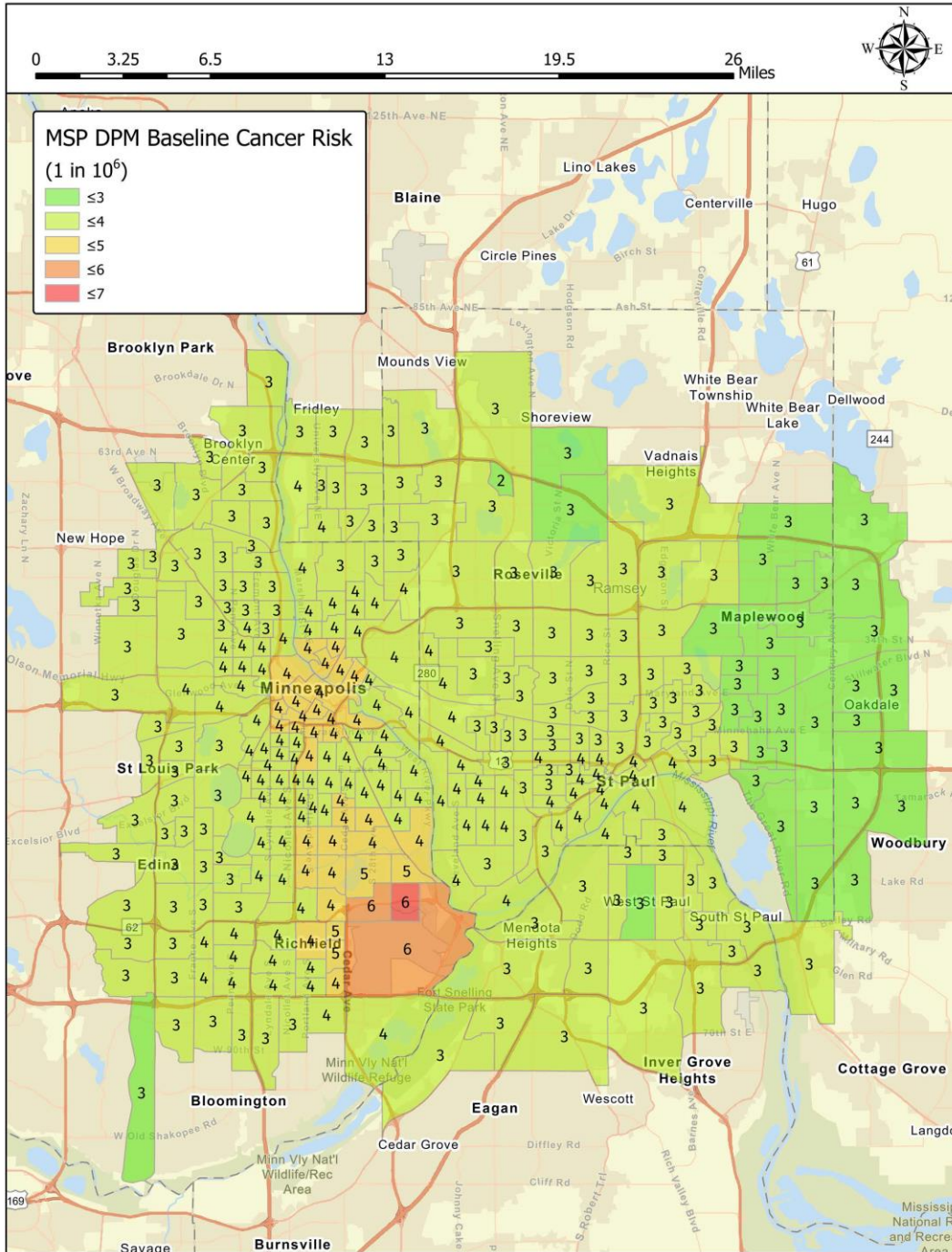
6.7.1.1 NATA Risk Data

Figure 6-67. Minneapolis/St. Paul Baseline NATA Total Cancer Risks



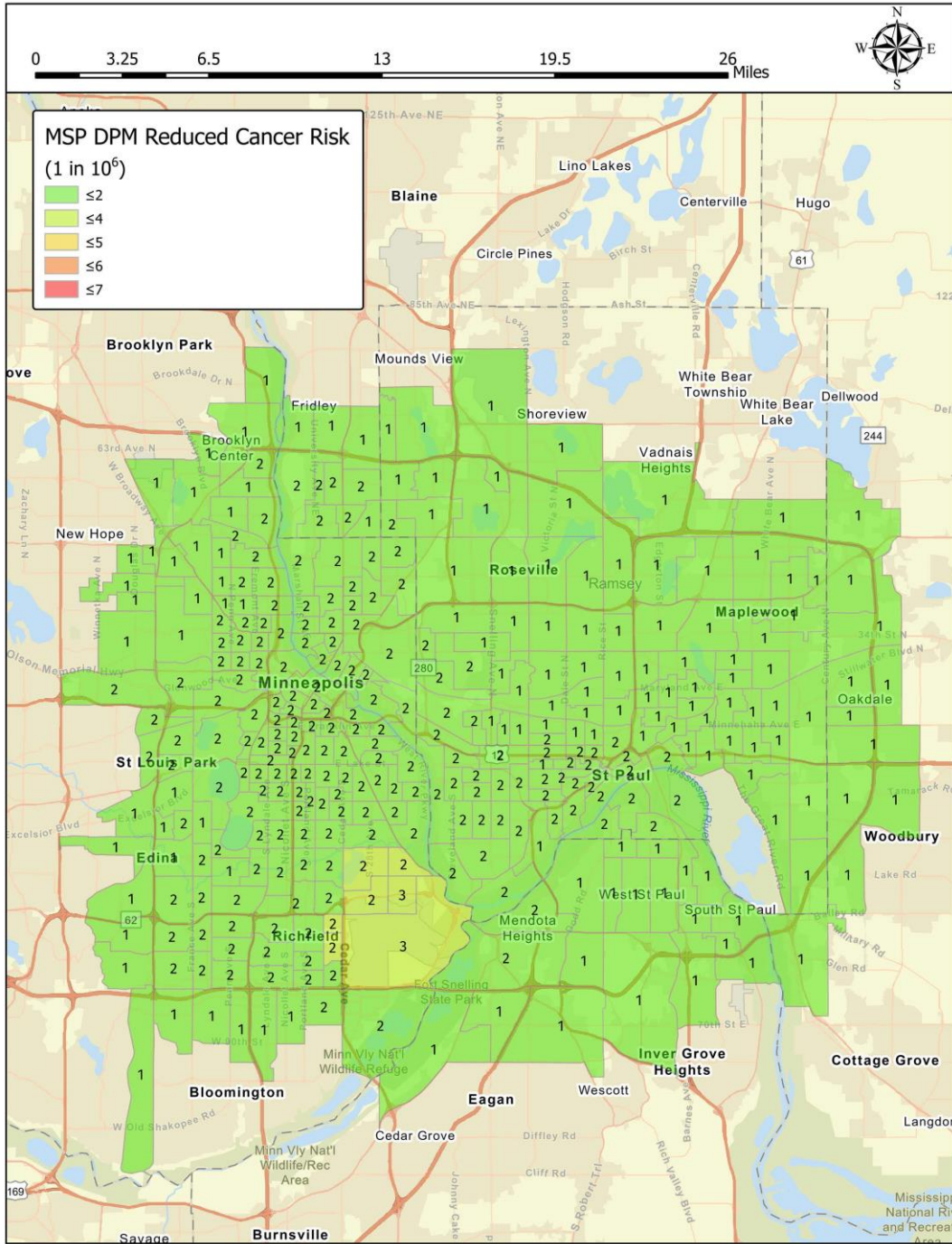
According to the NATA, the maximum baseline cancer risk in the Minneapolis/St. Paul community is 40.02 cancer cases per million residents for census tract 27123040801, with a population of 3,000 residents. When accounting for all of the communities assessed, the total cancer burden for the Minneapolis/St. Paul community is 36 cancer cases expected over a 70-year timeline among a total community population of 1,185,210.

Figure 6-68. Minneapolis/St. Paul Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the MSP community is 6 cancer cases per million residents for census tract 27053012102, with a population of 2,819 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the MSP community is 4 cancer cases expected over a 70-year timeline among a total community population of 1,185,210.

Figure 6-69. Minneapolis/St. Paul Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Minneapolis/St. Paul community becomes 3 cancer cases per million residents for census tract 27053012102, with a population of 2,819 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the MSP community becomes 2 cancer cases expected over a 70-year timeline among a total community population of 1,185,210.

6.7.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB's HARP program with OEHHA cancer unit risk** values. The data is outlined in the following order:

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with baseline and reduced cancer risks using **CARB's HARP program**.

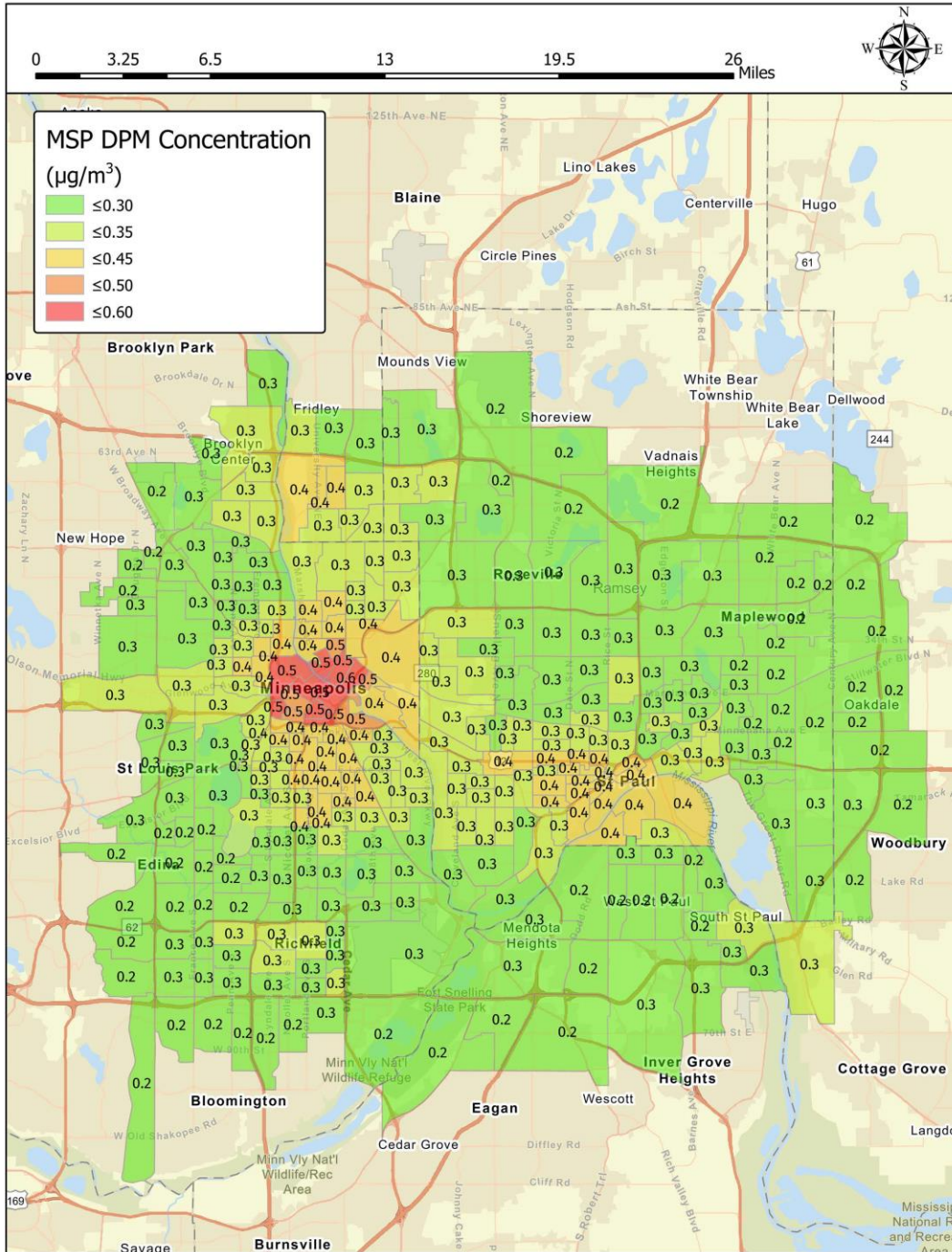
Figure 6-70 shows the baseline DPM concentrations provided by the NATA.

Figure 6-71 shows the baseline DPM-specific cancer risks as determined using the NATA concentration **values and CARB's HARP program**.

Figure 6-72 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the MSP community.

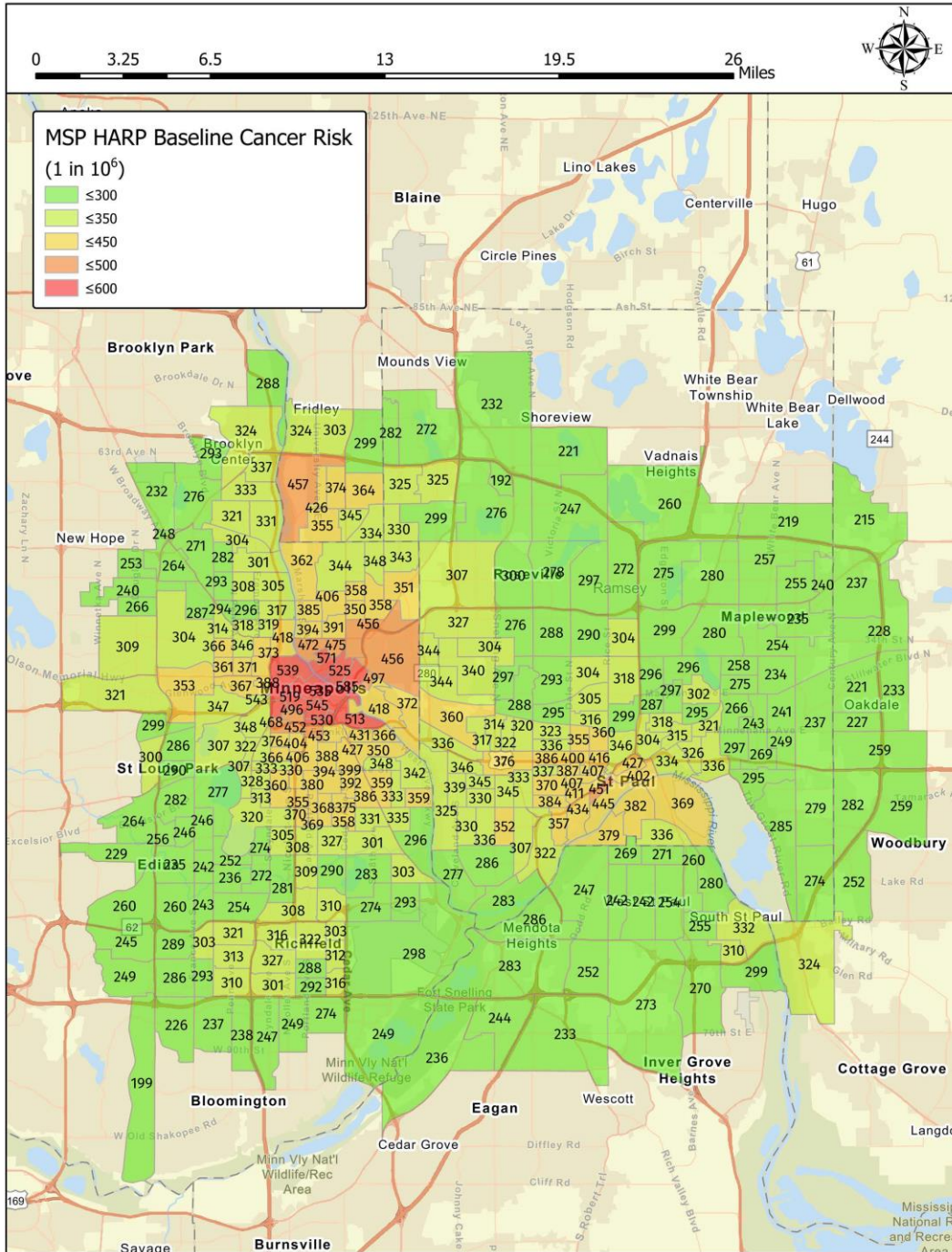
Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the MSP community.

Figure 6-70. Minneapolis/St. Paul Baseline NATA DPM Concentrations



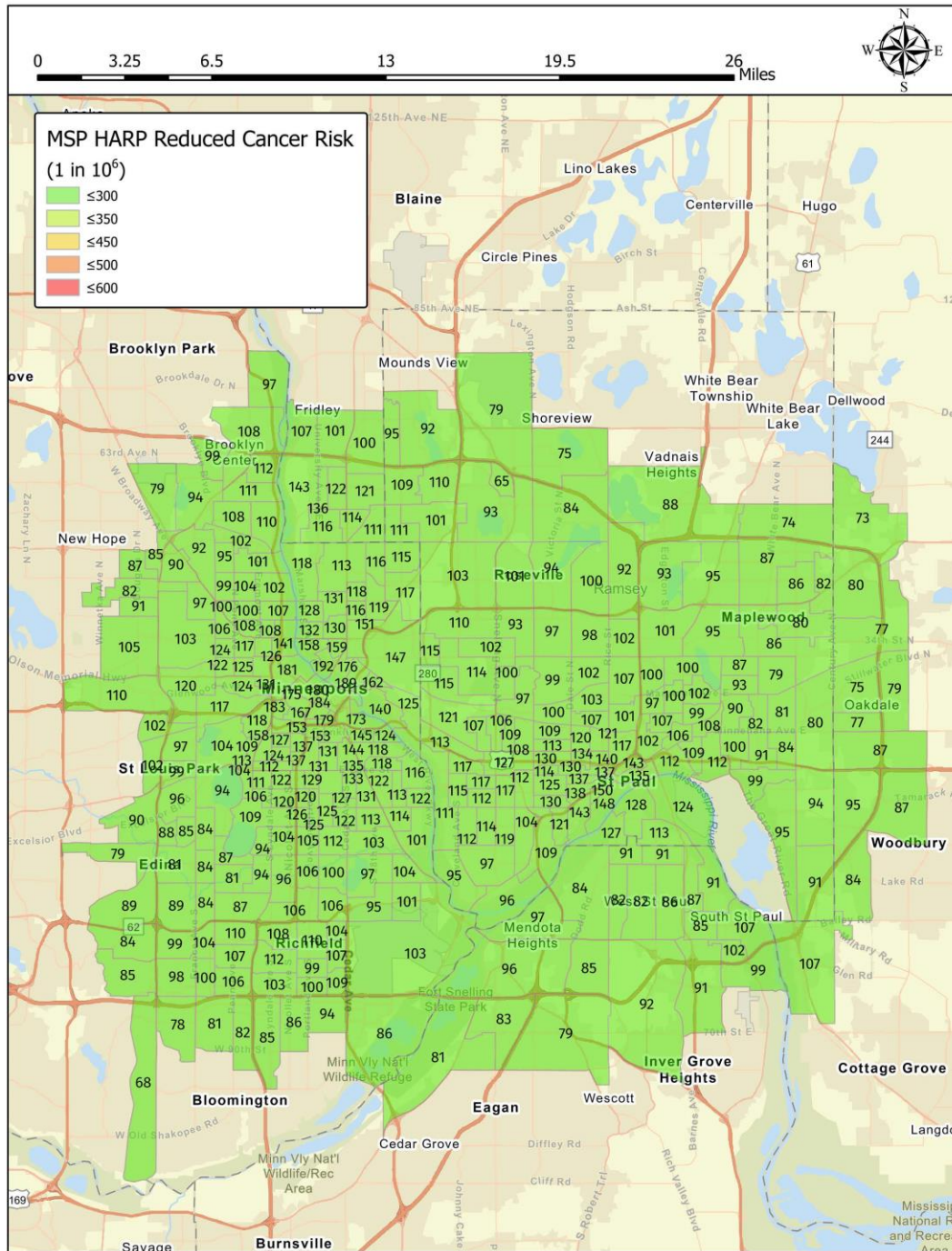
According to the NATA, the maximum baseline DPM concentration in the Minneapolis/St. Paul community is $0.56 \mu\text{g}/\text{m}^3$ for census tract 27053003800, with a population of 4,768 residents. The average DPM concentration of the MSP community is $0.31 \mu\text{g}/\text{m}^3$.

Figure 6-71. Minneapolis/St. Paul Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the Minneapolis/St. Paul community is 585 cancer cases per million residents for census tract 27053003800, with a population of 4,768 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Minneapolis/St. Paul community is 376 cancer cases expected over a 70-year timeline for a total community population of 1,185,210.

Figure 6-72. Minneapolis/St. Paul Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the MSP community becomes 192 cancer cases per million residents for census tract 27053003800, with a population of 2,084 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the MSP community becomes 127 cancer cases expected over a 70-year timeline among a total community population of 1,185,210.

6.7.2 Minneapolis/St. Paul Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Minneapolis/St. Paul. The following sources were utilized to generate the HRA.

- Minnesota State Department of Transportation – Traffic Counts (2019 Average Annual Daily Traffic)¹⁸

The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-13.

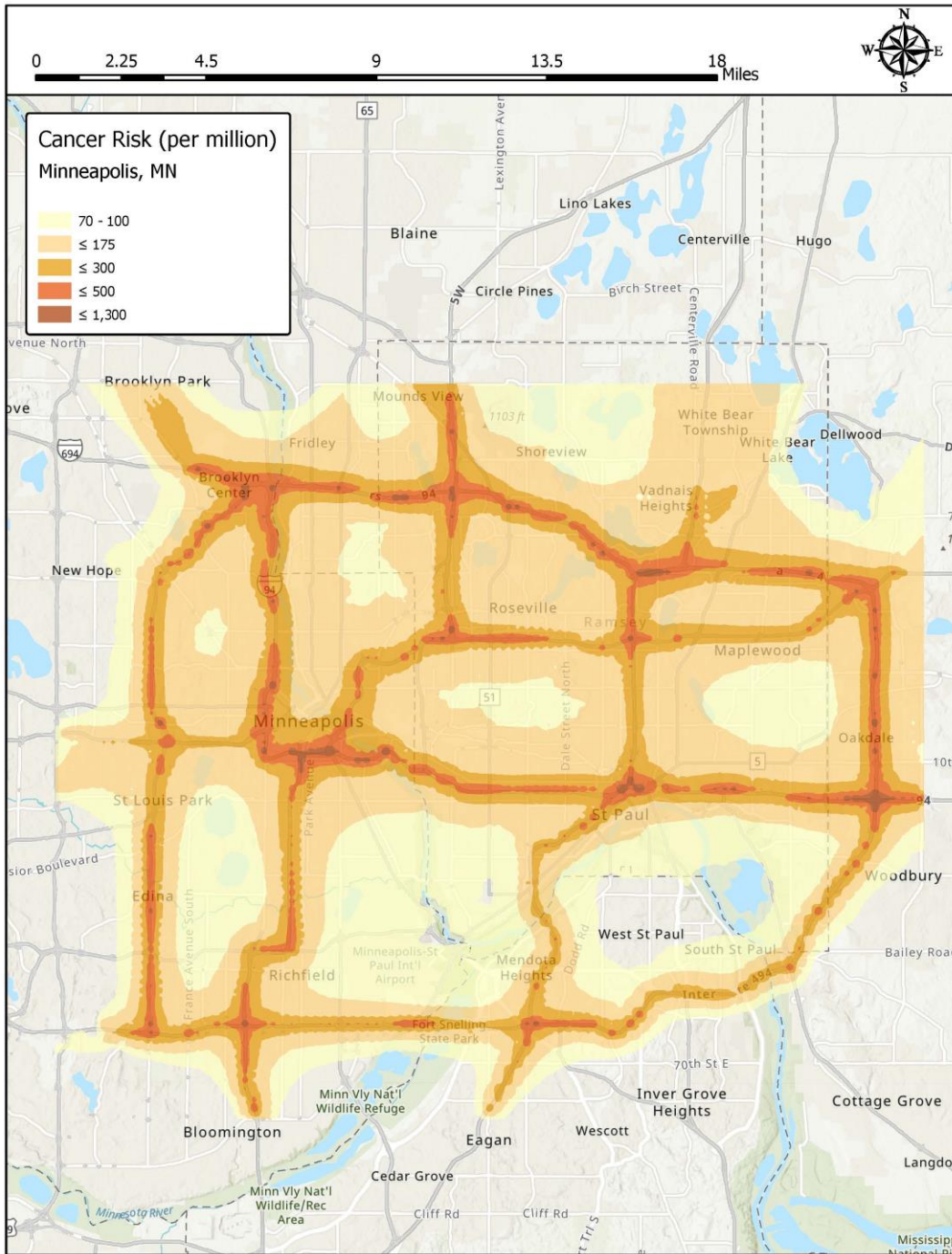
Table 6-13. Minneapolis/St. Paul Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of “Old Technology” Engine Emissions
I-494	I-494 – 112,313 AADT	27,171	59.7%
I-694	I-694 – 164,500 AADT	40,688	59.7%
35W	I-35 West – 120,000 AADT	28,752	59.7%
35E	I-35 East – 89,000 AADT	17,797	59.7%
I-394	I-394 – 57,133 AADT	3,232	59.7%
I-94	I-94 – 164,500 AADT	42,520	59.7%
MN100	Minnesota 100 – 127,500 AADT	20,251	59.7%
MN36	Minnesota 36 – 86,000 AADT	10,838	59.7%

These sources were modeled unit emission rates in AERMOD, and Table 6-13. listed emission rates were **input into CARB’s HARP software to determine cancer risks from the DPM concentrations determined by AERMOD**. While dispersion characteristics remained the same between baseline and reduced modeling scenarios, emission rates were reduced **according to the number of “old technology” engines combusting diesel**, based on source type. The table above shows the **Proportion of “Old Technology” Engine Emissions** where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

¹⁸ <https://dtdapps.coloradodot.info/otis/HighwayData>

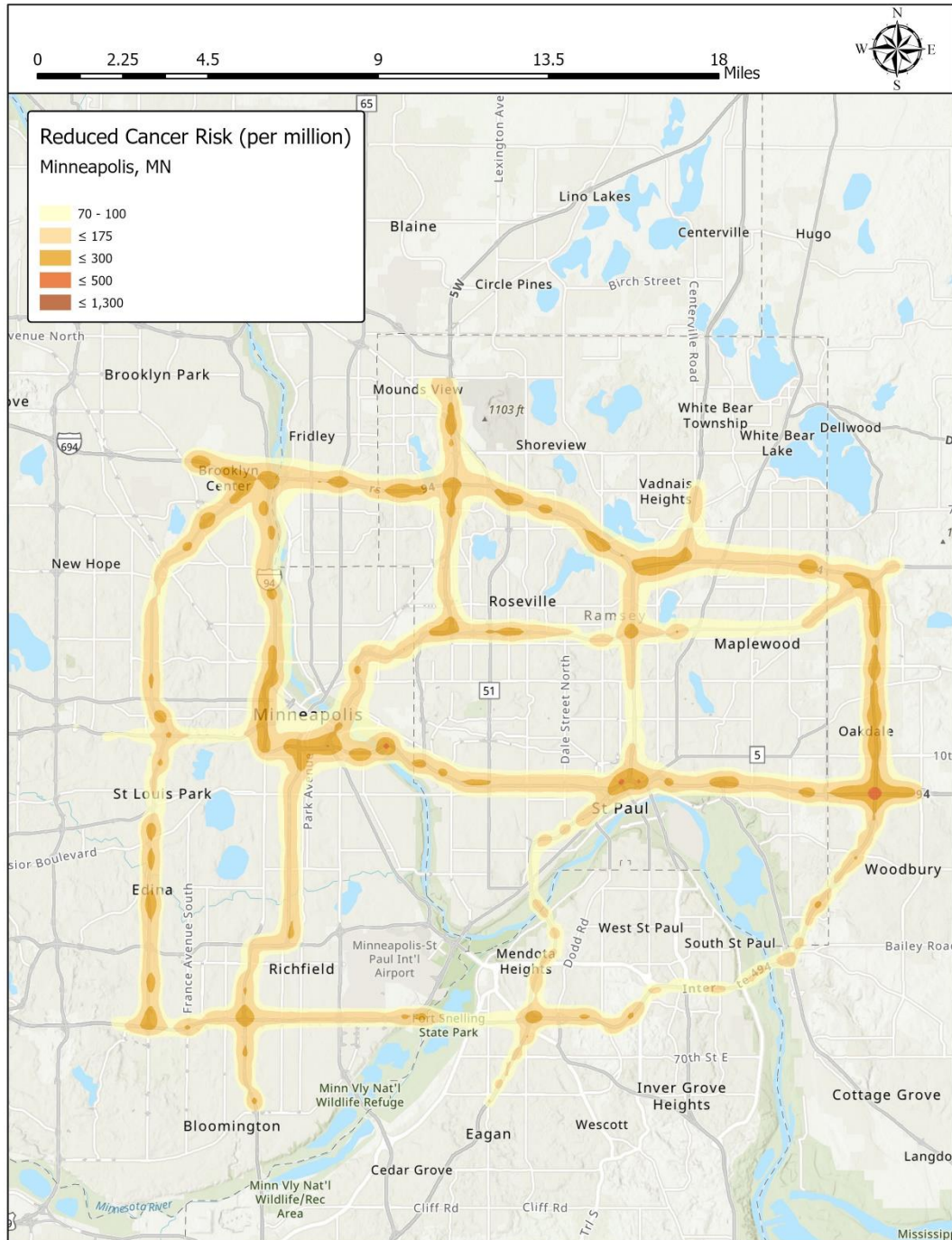
Figure 6-73. Minneapolis/St. Paul Baseline Health Risk Assessment Isoleths



The site-specific HRA shows that the point of maximum impact (PMI) is higher than the NATA/HARP evaluation, with an impact of 981 cancer cases per million residents. This PMI does not occur at a residential receptor, though, and does not represent an actual risk to residences in the area. The MEIR occurs at 477,217 m E, and 4,979,349.8 m N (NAD 83, UTM Zone 17), with a baseline risk of 684 cancer cases per million residents. This MEIR is higher than the NATA/HARP hybrid risks evaluated for that census tract (27163070910) with a total risk of 259 in a million. This HRA does not capture all of the cancer-causing

sources in the area but does demonstrate that NATA values are in-line with the site-specific demonstration with some extremely high local maxima due to local residences proximity to highways.

Figure 6-74. Minneapolis/St. Paul Reduced Health Risk Assessment Isoleths



The reduced cancer risk at the PMI and MEIR is 395 and 276 in 1 million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 408 in 1 million at the MEIR.

6.7.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-14 below.

Table 6-14. Minneapolis/St. Paul Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	49.7	\$1,578,445
Asthma Symptoms - Albuterol use	7,862	\$2,717
ER visits - All Cardiac Outcomes	4.9	\$5,695
ER visits – Respiratory	10.9	\$9,552
HA – All – Respiratory	2.1	\$30,660
HA – Alzheimer’s Disease	12.6	\$155,721
HA – Cardio Cerebro- and Peripheral Vascular Disease	2.3	\$35,920
HA – Parkinson’s Disease	1.4	\$17,953
HA – Respiratory-2	0.3	\$0
HA – Respiratory-2 HA – All Respiratory	2.4	\$0
Incidence – Asthma	61.0	\$2,724,884
Incidence – Hay Fever/Rhinitis	377.2	\$226,256
Incidence – Lung Cancer	2.9	\$37,090
Incidence – Out of Hospital Cardiac Arrest	0.3	\$12,297
Incidence – Stroke	1.2	\$40,653
Minor Restricted Activity Days	17,790	\$1,237,825
Mortality – All Cause	19.1	\$149,430,336
Work Loss Days	3,027	\$614,206
Total		\$156,160,209

6.8 Las Vegas, Nevada

6.8.1 NATA Health Risks

The subsections below review the NATA data available for the Las Vegas, NV (Las Vegas) community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-75 shows the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

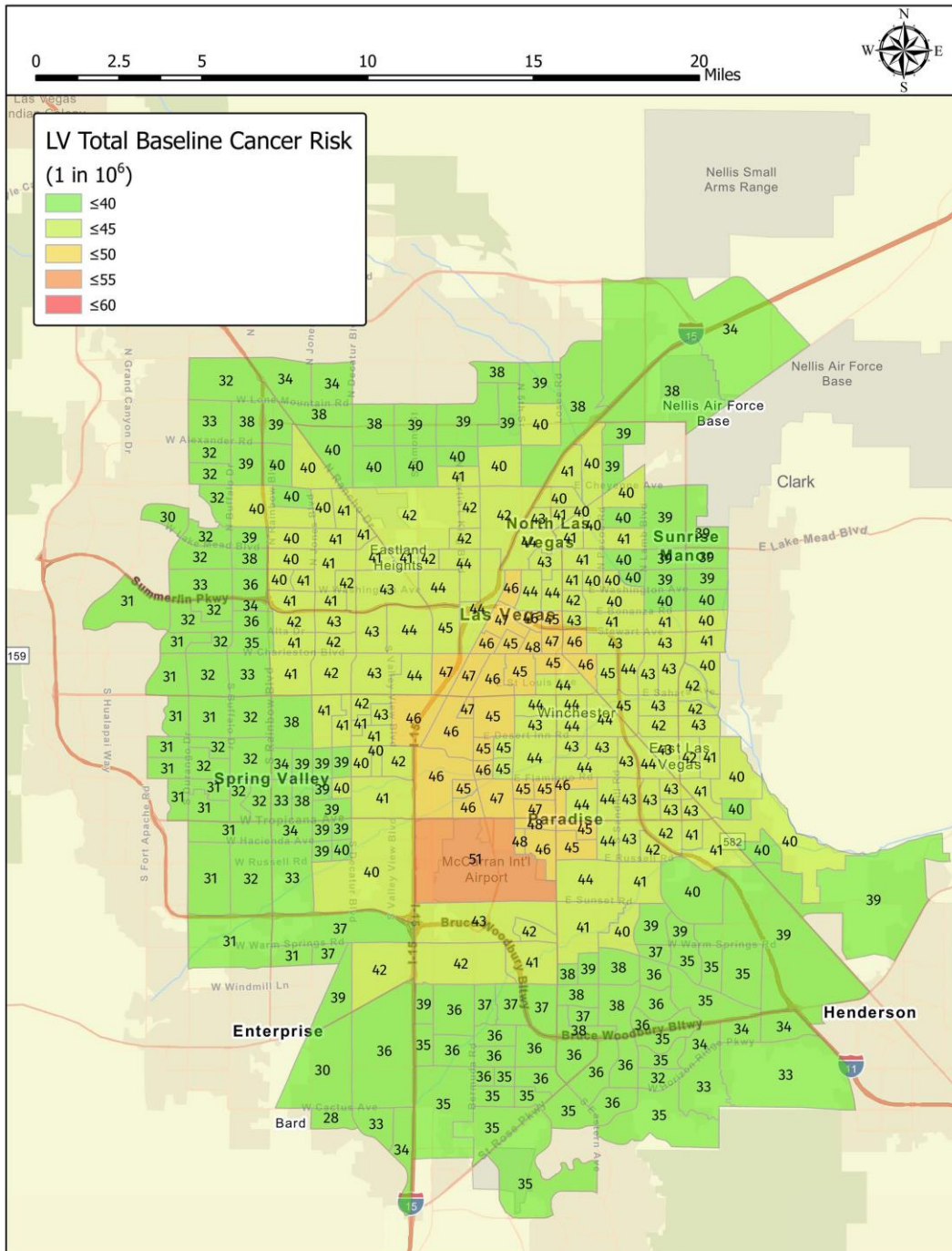
Figure 6-76 shows those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-77 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Las Vegas community.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the Las Vegas community.

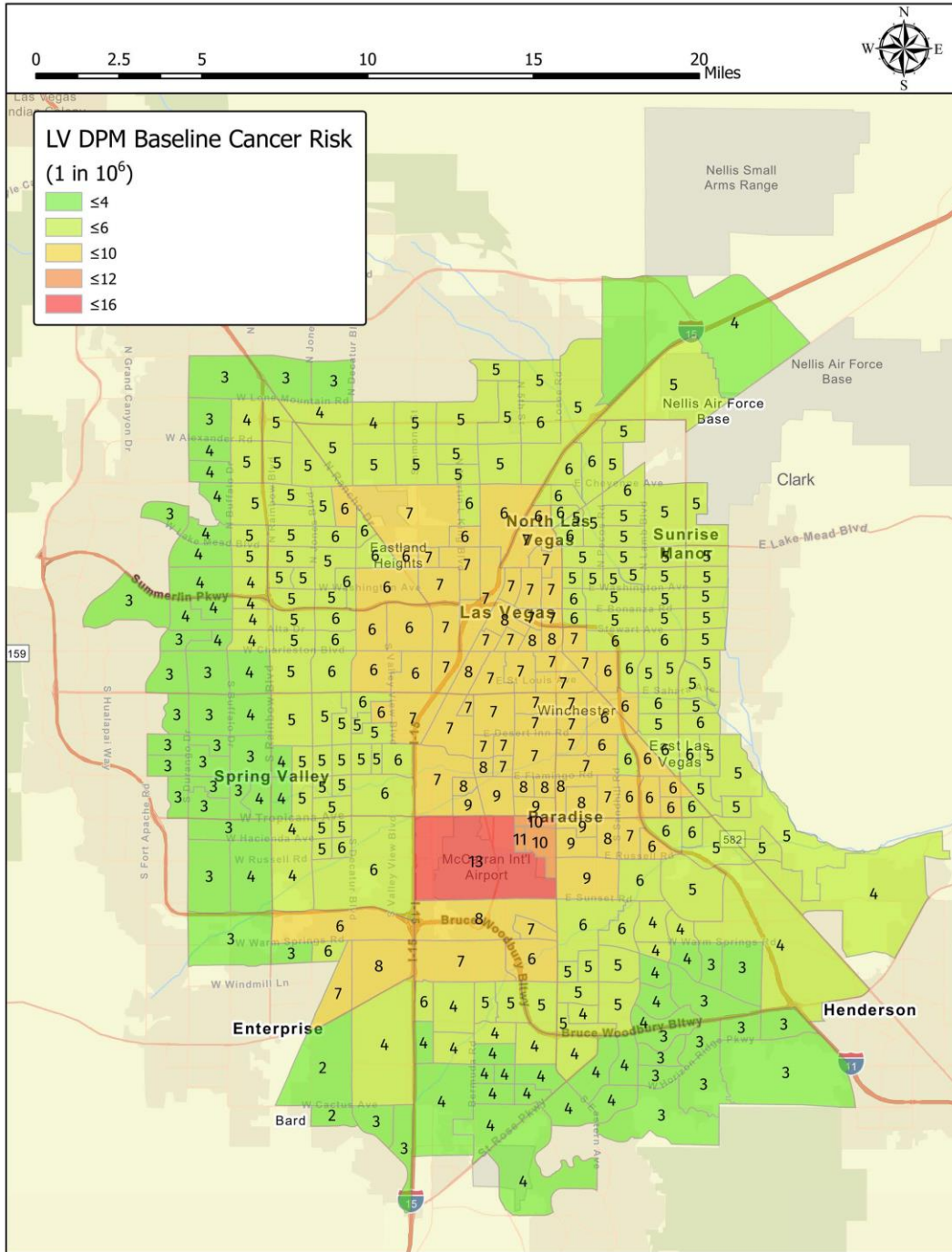
6.8.1.1 NATA Risk Data

Figure 6-75. Las Vegas Baseline NATA Total Cancer Risks



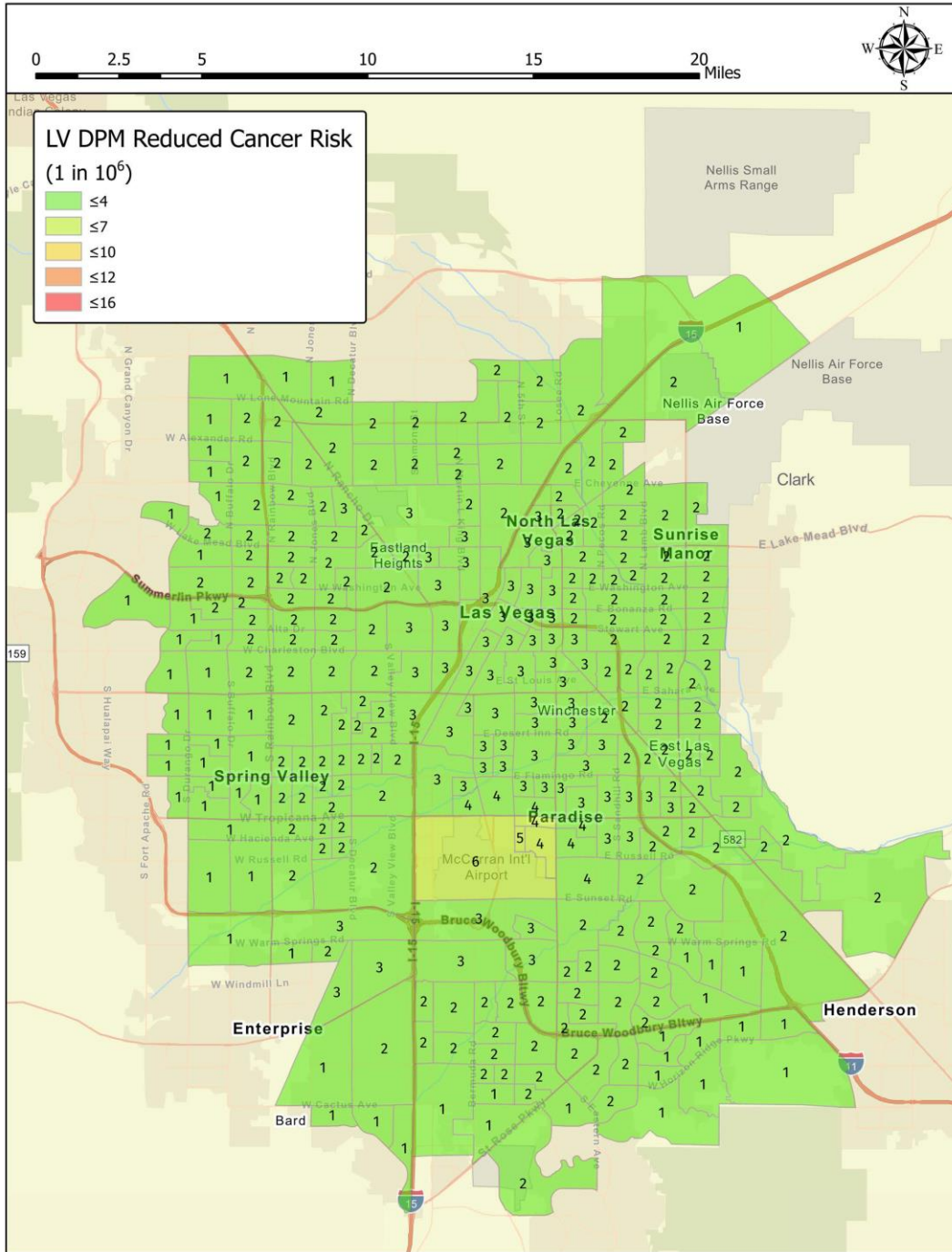
According to the NATA, the maximum baseline cancer risk in the Las Vegas community is 50.73 cancer cases per million residents for census tract 32003006800, with a population of 4,543 residents. When accounting for all of the communities assessed, the total cancer burden for the Las Vegas community is 50 cancer cases expected over a 70-year timeline among a total community population of 1,262,051.

Figure 6-76. Las Vegas Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the Las Vegas community is 13 cancer cases per million residents for census tract 32003006800, with a population of 4,543 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Las Vegas community is 7 cancer cases expected over a 70-year timeline among a total community population of 1,262,051.

Figure 6-77. Las Vegas Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Las Vegas community becomes 6 cancer cases per million residents for census tract 32003006800, with a population of 4,543 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Las Vegas community becomes 3 cancer cases expected over a 70-year timeline among a total community population of 1,262,051.

6.8.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB's HARP program with OEHHA cancer unit risk values. The data is outlined in the following order:**

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program.**

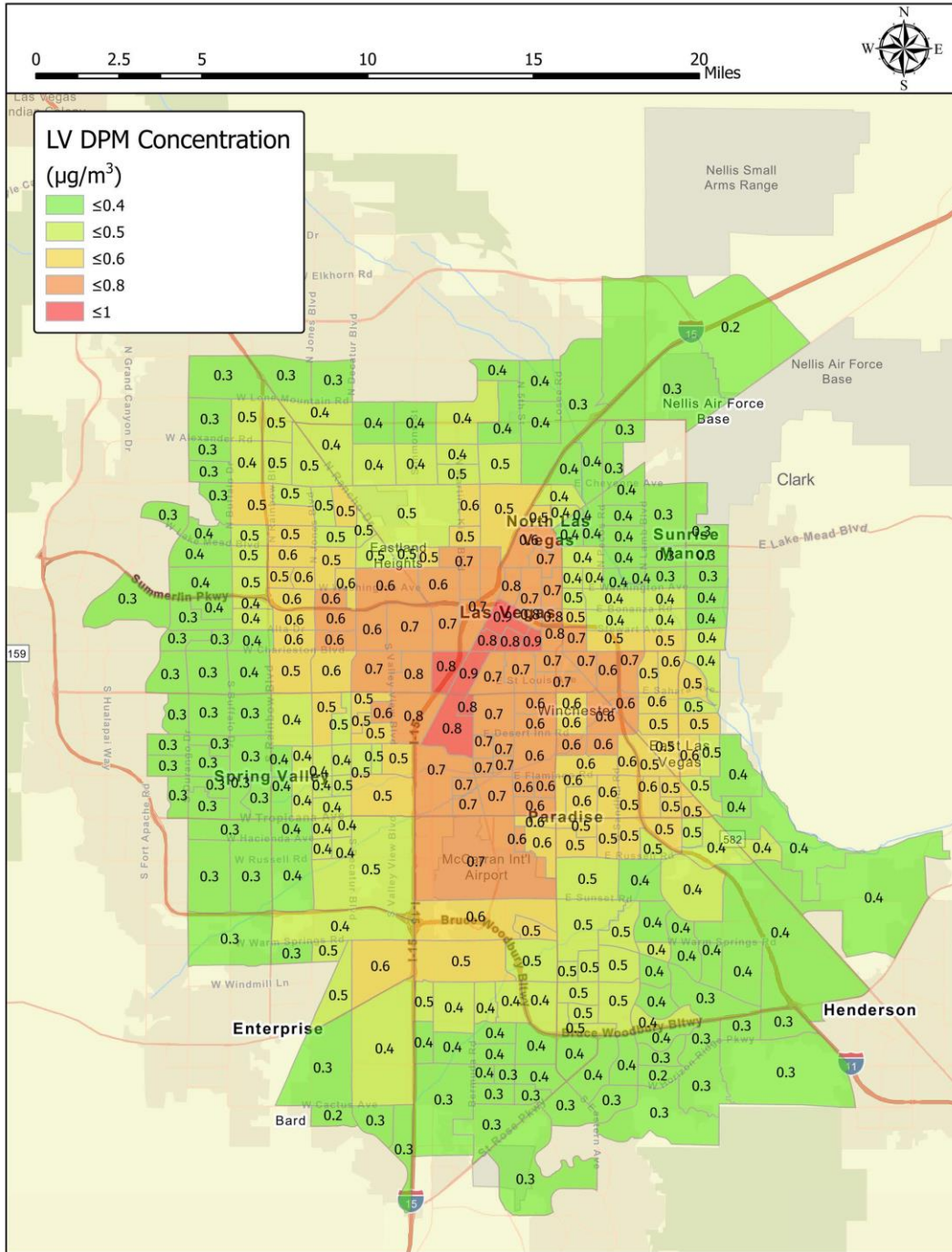
Figure 6-78 shows the baseline DPM concentrations provided by the NATA.

Figure 6-79 shows the baseline DPM-specific cancer risks as determined using the NATA concentration **values and CARB's HARP program.**

Figure 6-80 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Las Vegas community.

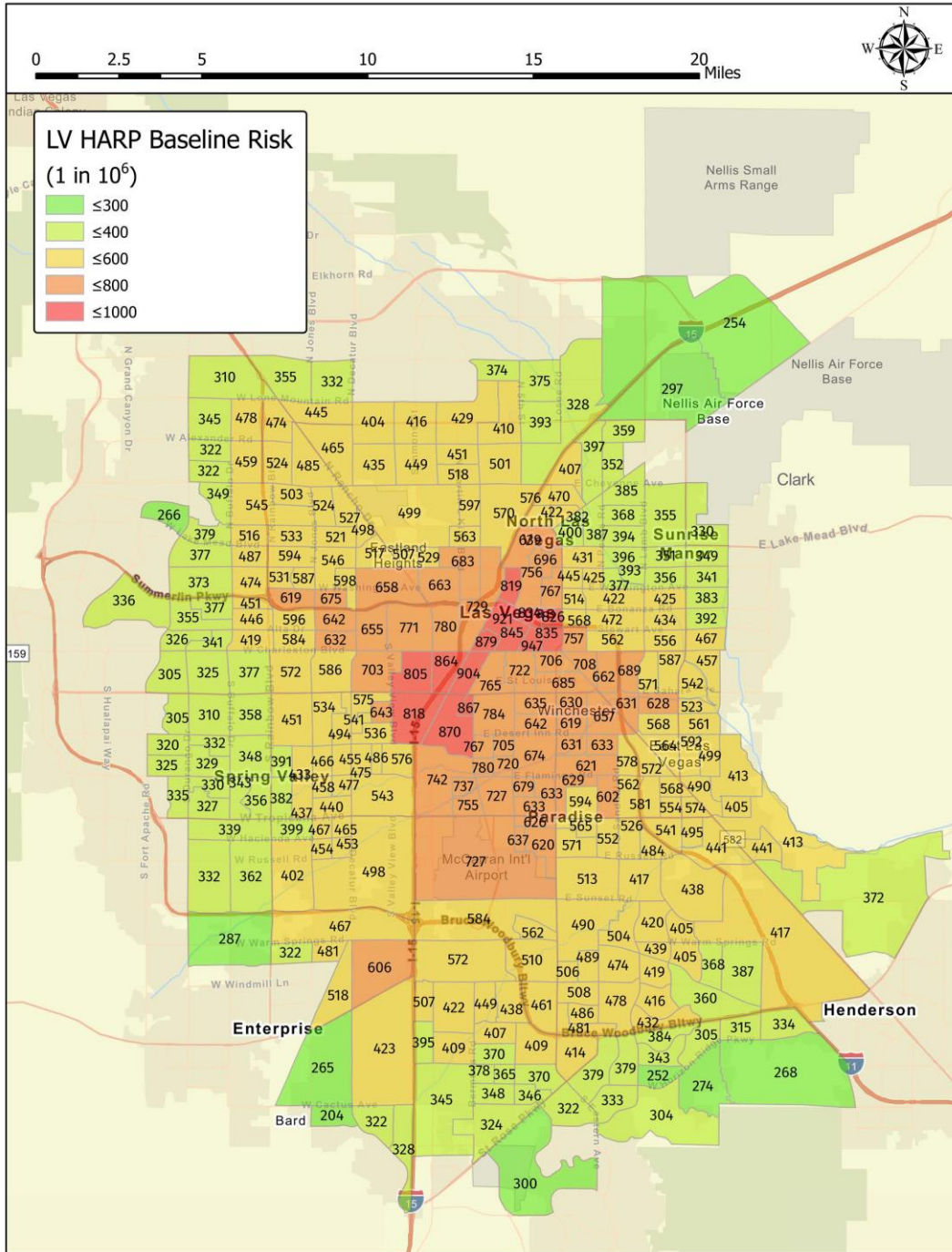
Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the Las Vegas community.

Figure 6-78. Las Vegas Baseline NATA DPM Concentrations



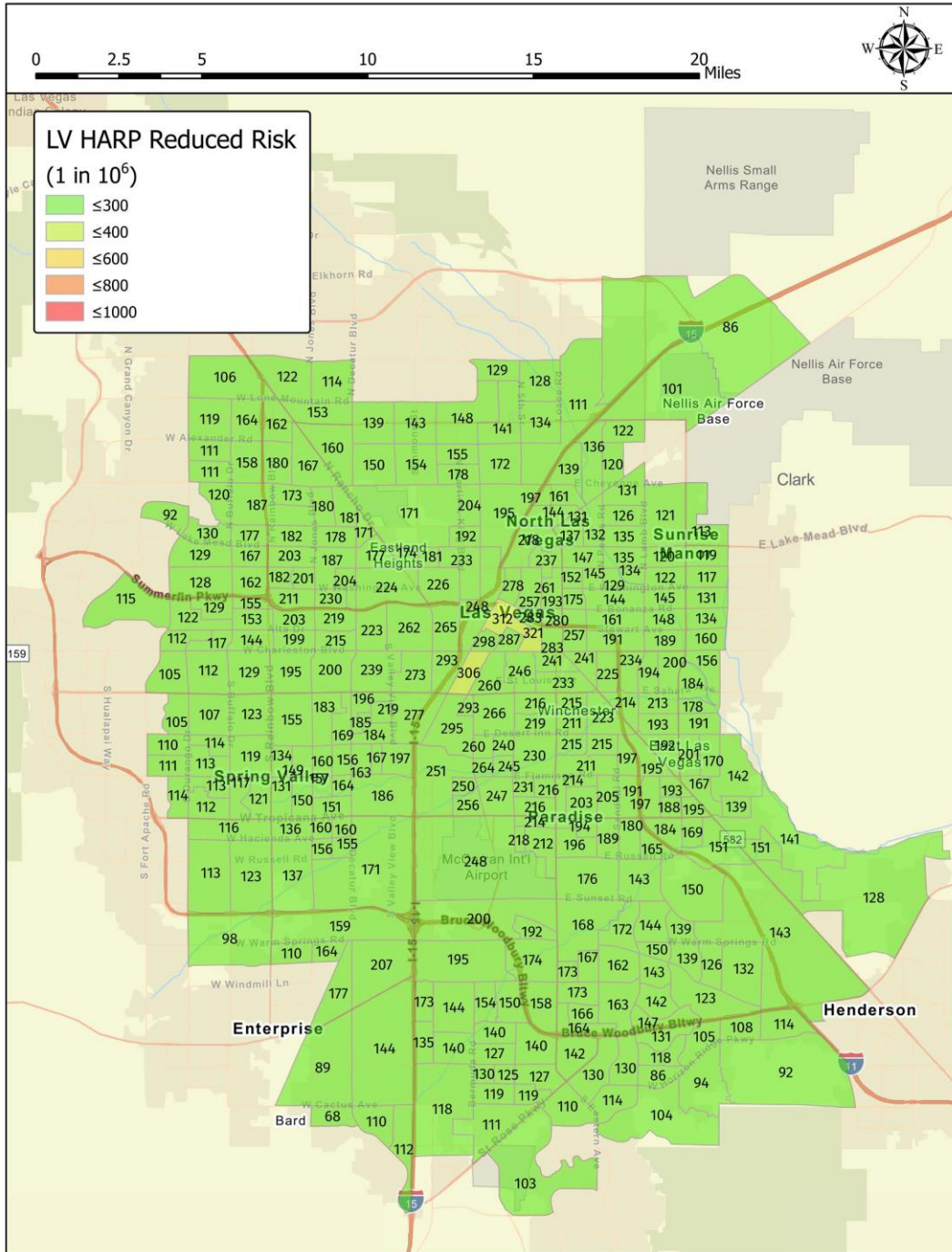
According to the NATA, the maximum baseline DPM concentration in the Las Vegas community is 0.90 $\mu\text{g}/\text{m}^3$ for census tract 32003000600, with a population of 2,824 residents. The average DPM concentration of the Las Vegas community is 0.47 $\mu\text{g}/\text{m}^3$.

Figure 6-79. Las Vegas Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the Las Vegas community is 947 cancer cases per million residents for census tract 32003000600, with a population of 2,824 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Las Vegas community is 612 cancer cases expected over a 70-year timeline among a total community population of 1,262,051.

Figure 6-80. Las Vegas Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Las Vegas community becomes 321 cancer cases per million residents for census tract 32003000600, with a population of 2,824 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Las Vegas community becomes 209 cancer cases expected over a 70-year timeline among a total community population of 1,262,051.

6.8.2 Las Vegas Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Las Vegas. The following sources were utilized to generate the HRA.

- Nevada State Department of Transportation – Traffic Counts (2019 Average Annual Daily Traffic)¹⁹

The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-15.

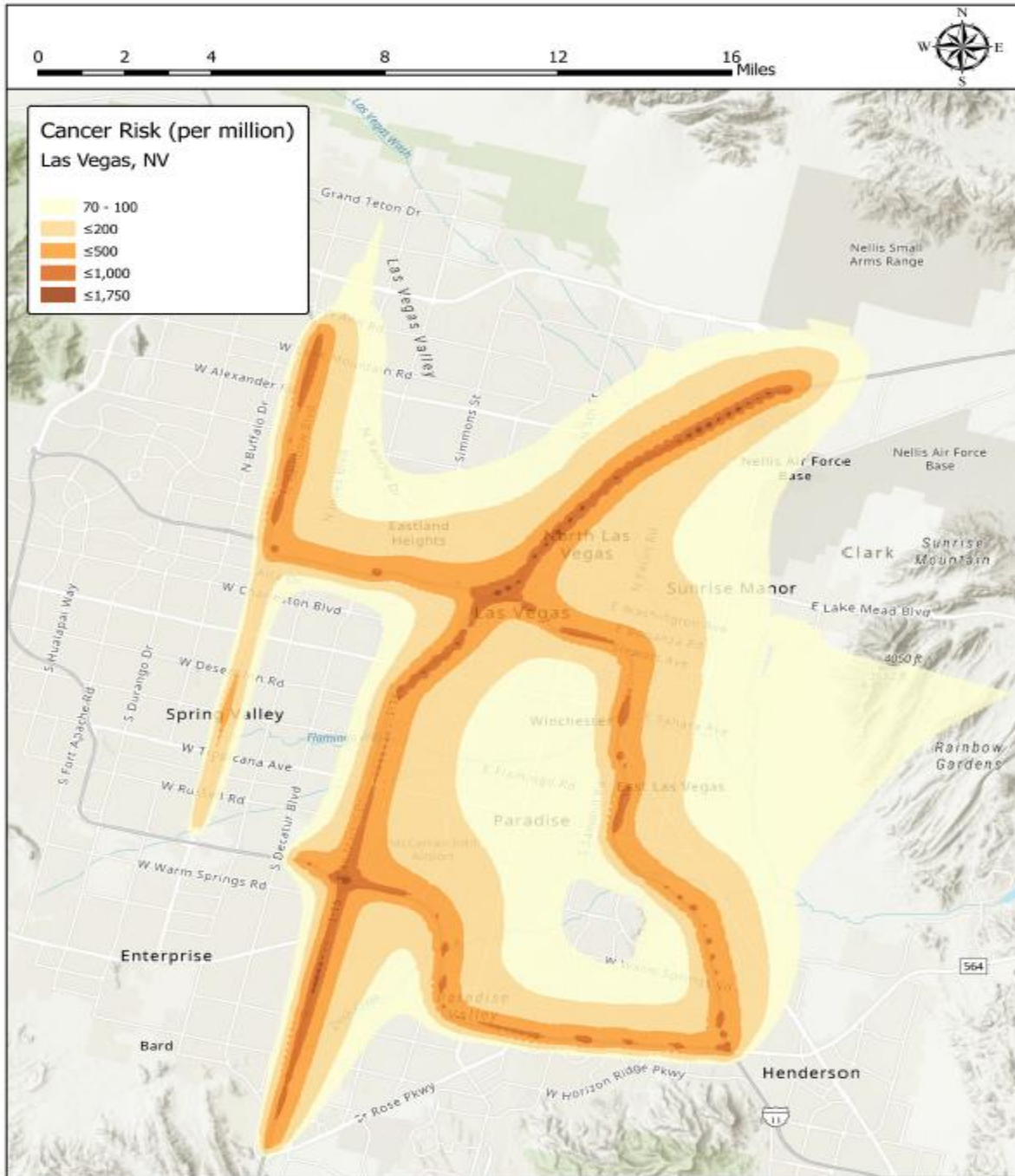
Table 6-15. Las Vegas Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of “Old Technology” Engine Emissions
15	State Route 15 – 185,441 AADT	44,235	59.7%
95	State Route 95 – 54,571 AADT	4,068	59.7%
215	State Route 215 – 171,900 AADT	21,260	59.7%
599/595/573	State Route 599/595/573 – 174,475 AADT	43,333	59.7%

These sources were modeled unit emission rates in AERMOD, and Table 6-15 listed emission rates were input into CARB’s HARP software to determine cancer risks from the DPM concentrations determined by AERMOD. While dispersion characteristics remained the same between baseline and reduced modeling scenarios, emission rates were reduced according to the number of “old technology” engines combusting diesel, based on source type. The table above shows the Proportion of “Old Technology” Engine Emissions where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

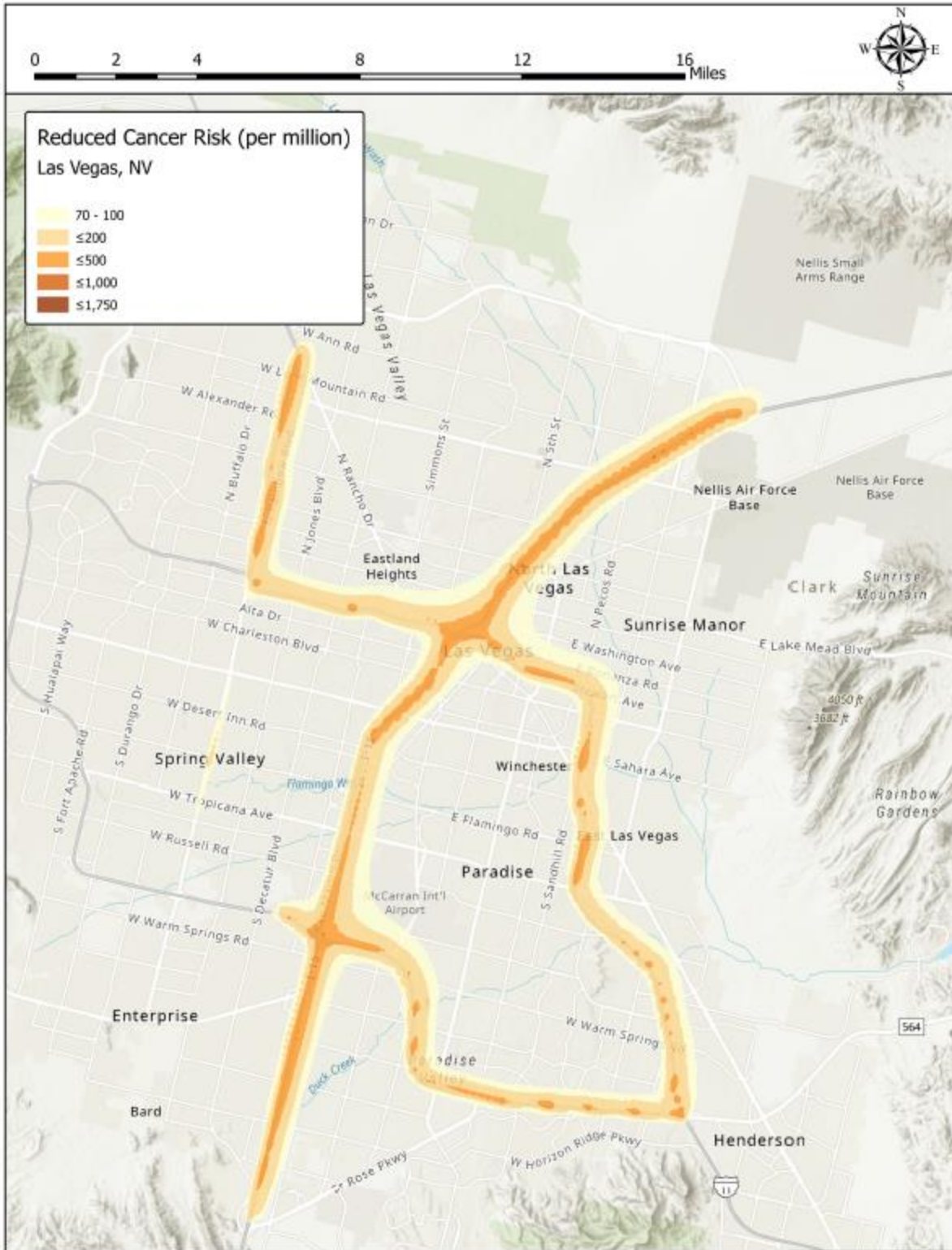
¹⁹ <https://ndot.maps.arcgis.com/apps/webappviewer/index.html?id=278339b4605e4dda8da9bddd2fd9f1e9>

Figure 6-81. Las Vegas Baseline Health Risk Assessment Isopleths



The site-specific HRA shows that the point of maximum impact (PMI) is substantially higher than the NATA/HARP evaluation, with an impact of 1,746 cancer cases per million residents. This PMI does not occur at a residential receptor, though, and does not represent an actual risk to residences in the area. The MEIR occurs at 657,581.7 m E, and 4,012,698.6 m N (NAD 83, UTM Zone 11), with a baseline risk of 1,194 cancer cases per million residents. This MEIR is higher than the NATA/HARP hybrid risks evaluated for that census tract (32003003409) with a total risk of 474 in a million. This HRA does not capture all of the cancer-causing sources in the area but does demonstrate that NATA values are in-line with the site-specific demonstration with some extremely high local maxima due to local residences proximity to highways.

Figure 6-82. Las Vegas Reduced Health Risk Assessment Isopleths



The reduced cancer risk at the PMI and MEIR is 703 and 481 in 1 million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 713 in 1 million at the MEIR.

6.8.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using USEPA's BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-16 below.

Table 6-16. Las Vegas Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	65.4	\$2,124,938
Asthma Symptoms - Albuterol use	8,817	\$3,047
ER visits - All Cardiac Outcomes	7.2	\$8,397
ER visits – Respiratory	13.7	\$11,939
HA – All – Respiratory	1.6	\$30,491
HA – Alzheimer's Disease	7.3	\$87,421
HA – Cardio Cerebro- and Peripheral Vascular Disease	2.8	\$43,487
HA – Parkinson's Disease	1.2	\$15,915
HA – Respiratory-2	0.4	\$0
HA – Respiratory-2 HA – All Respiratory	2.0	\$0
Incidence – Asthma	69.3	\$3,095,512
Incidence – Hay Fever/Rhinitis	428.6	\$257,112
Incidence – Lung Cancer	3.5	\$44,684
Incidence – Out of Hospital Cardiac Arrest	0.4	\$15,071
Incidence – Stroke	1.5	\$50,797
Minor Restricted Activity Days	21,667	\$1,507,632
Mortality – All Cause	24.8	\$194,057,115
Work Loss Days	3,681	\$555,735
Total		\$201,909,293

6.9 Buffalo, New York

Distillate oil is widely used in many northeastern states to supply heat to homes. The individual chemical components generated from the combustion of residential heating oil have associated toxicity factors, and have been analyzed for associated health risks by the U.S. EPA. The following sections identify how the U.S. EPA and state agencies have reported on distillate oil combustion for residential heating and how the associated health risks are determined by the U.S. EPA.

6.9.1 NATA Health Risks

U.S. EPA determines county-wide health risks from residential heating oil combustion by quantifying the emissions of stationary nonpoint sources as part of the National Emission Inventory (NEI) dataset, which is subsequently evaluated in the National Air Toxics Assessment (NATA).

According to the 2014 NEI Technical Support Document (TSD)²⁰ in Table 4-70, non-wood residential heating by combustion of distillate oil was reported by some state, local, and tribal agencies. EPA estimates were also available for these sources. Table 4-71 of the TSD outlines the agencies that reported data for non-wood residential heating emissions. For any state, local, or tribal agency that did not provide non-wood residential heating data, EPA estimates were utilized instead. According to the NEI TSD:

*The general approach to calculating emissions for all fuel types is to take state-level fuel-specific (natural gas, distillate oil, kerosene, coal, and LPG) consumption from the EIA and allocate it to the **county level**. ... County-level fuel consumption is multiplied by the emission factors to calculate emissions.²¹*

Fuel type consumption by energy use sector was obtained from the State Energy Data System (SEDS) 2014 Consumption tables published by the U.S. Energy Information Administration (EIA). Distillate oil consumption is represented in the SEDS table by the Data Series Name (DSN). State-level fuel type **consumption was allocated to each county using the U.S. Census Bureau's 2014 5-year** estimate from its Census Detailed Housing report. These data include the number of housing units using a specific type of fuel for residential heating. State fuel type consumption was allocated to each county using the ratio of the number of houses burning natural gas, distillate oil, kerosene, or LPG in each county to the total number of houses burning natural gas, distillate oil, kerosene, or LPG in the state.

The NATA TSD states that the reported Hazardous Air Pollutant (HAP) emissions from the nonpoint 2014 NEI sources were modeled using a hybrid approach with the Community Multiscale Air Quality (CMAQ) and AERMOD models for the 52 most prevalent and high-risk toxics. Coarse, region-wide impacts were determined on a county level using 12-kilometer grids in the CMAQ model. AERMOD was utilized to generate near-field concentrations using gridded receptors (1 km in highly populated areas [>1 million population], 4 km in other areas), census block centroid receptors, and monitoring site receptors. These

²⁰ https://www.epa.gov/sites/production/files/2018-07/documents/nei2014v2_tsd_05jul2018.pdf

²¹ The NEI TSD uses standard USEPA toxic emission factor datasets for distillate oil combustion. The Northeast States for Coordinated Air Use Management (NESCAUM) completed analyses in 2010 and 2015 to determine levels of sulfur and 10 trace elements in distillate and residual oil samples collected from terminals in New York City, Albany, and eastern Massachusetts (*Updated Determination of Sulfur and Other Trace Element Content of Fuel Oil in New York State*, NYSERDA Report Number 17-11; 2017). Using the NESCAUM survey results, the arsenic and nickel-based cancer risks may differ because of the differences in levels of those metals found by NESCAUM relative to the USEPA dataset, but the Valuation of Health Benefits results **using USEPA's Ben-MAP** model would remain unchanged since these results are dependent only on reduction of very fine particulate matter (PM_{2.5}).

results were then weighted according to grid cell averages to determine census block and tract exposures for the 52 toxics. All other toxics were modeled directly using AERMOD.

Table 4-71 of the NEI TSD states that the New York State Department of Environmental Conservation provided 100% of the residential fuel oil combustion data to EPA for the NEI. Given the population density of Buffalo, NY, the 1 km grid cells were utilized in NATA

6.9.2 Toxicity of Heating Oil Combustion Compounds

Table 6-17 below lists the U.S. EPA and CARB toxicity “slope” factors for air toxic compounds released by combustion of residential heating oil. In general, the CARB toxicity factors are higher for all of the distillate oil combustion compounds, with the exception of Arsenic and Formaldehyde. Specifically, Hexavalent Chromium has the highest toxicity slope factor of all the pollutants and is 12.5 times higher than the equivalent EPA slope factor. Therefore, the use of CARB’s HARP program represents a high-end of health risks from residential heating oil combustion.

Table 6-17. Comparison of U.S. EPA and CARB Heating Oil Exhaust Toxicity Values

CAS	Pollutant	CARB Slope Factor (1/μg/m ³)	EPA Slope Factor (1/μg/m ³)	CARB vs EPA Value
75070	Acetaldehyde	0.000027	0.000022	123%
7440382	Arsenic	0.0033	0.0043	77%
56553	B[a]anthracene	0.00011	0.00006	183%
71432	Benzene	0.000029	0.0000078	372%
7440417	Beryllium	0.0024	0.0024	100%
7440439	Cadmium	0.0042	0.0018	233%
218019	Chrysene	0.000011	0.0000006	1833%
18540299	Cr(VI)	0.15	0.012	1250%
53703	D[a,h]anthracene	0.0012	0.0006	200%
50000	Formaldehyde	0.000006	0.000013	46%
193395	In[1,2,3-cd]pyr	0.00011	0.00006	183%
7439921	Lead	0.000012	0	N/A
91203	Naphthalene	0.000034	0.000034	100%
7440020	Nickel	0.00026	0.00024	108%

6.9.3 Data Sources and Emissions Inventories

In order to determine the heating oil usage in a given area, the heating oil use was first determined from the NEI data on a county-wide basis. The NEI data provides annual emissions from various source categories, and hence, the PM_{2.5} annual emissions from the home heating oil combustion category were

used to back-calculate the annual heating oil usage using the PM_{2.5} emission factor for home heating oil combustion (units of lb/1,000 gallons).²²

Trinity identified areas that had high usages of heating oil utilizing U.S. Census data which tracks total number of homes that use heating oil within a given census tract.²³ Trinity identified census tract 68 as having the highest heating oil use in 2020. The amount of heating oil used for that year was multiplied by the emission factors from the 2017 NEI to calculate PM_{2.5} and toxic emission rates as follows:²⁴

$$\text{Heating Oil Usage} \left(\frac{\text{gallons}}{\text{year}} \right) \times \text{NEI Emission Factor} \left(\frac{\text{lb}}{1,000 \text{ gal}} \right) \times \frac{1 \text{ year}}{8,760 \text{ hours}} = \text{Emission Rate} \left(\frac{\text{lbs}}{\text{hour}} \right)$$

6.9.4 Models

Ambient concentrations from the above sources were determined using USEPA’s approved AERMOD

Modeling System. The model utilized local surface and upper air meteorological data processed using EPA AERMINUTE and AERMET meteorological data processors, along with preprocessed terrain data prepared by the United States Geological Survey. To provide a refined analysis of local maxima, the emission sources are modeled as a polygon area source with the dimensions of the census tract modeled. However, due to the geographic scale of the modeling domain, a simplified approach was utilized (e.g., buildings and structures will not be independently modeled).

Table 6-18 below indicates the source parameters used for Buffalo census tract 68.

Table 6-18. Buffalo Census Tract 68 Modeling Parameters

Source ID	X (m)	Y (m)	Elevation (m)	Emission Rate (g/m ² -s)	Release Height (m)	Initial Vert. Dimension (m)
AREAPOLY	183,082.7	4,757,278.6	198.34	1.153E-06	0	0

Given that most of the sources in the Buffalo census tract are from single-story residences, a 0-foot release height was utilized for the polygon area source (12 vertices) with no initial vertical dimension. This does not account for buoyancy from plume rise, but it is consistent with EPA NATA modeling procedures of this source type.

The emission rate for the polygon area source is equal to one over the area of the polygon, representing a unit emission rate of 1 g/s. This unit emission rate was run for 8,760 hours, and the resulting period file concentration, representing the average concentration across all receptors over the 5-years of meteorological data, was used to generate ground level concentrations for each individual pollutant. The ground level concentrations and associated health risk impacts were determined using CARB’s HARP tool, which implements the OEHHA risk analysis methodology. Risk was determined in terms of excess cancer risk above baseline values due to distillate oil combustion.

²² See document titled “Residential Heating NEMO 2017 FINAL_4-2 update” located at: ftp://newftp.epa.gov/air/nei/2017/doc/supporting_data/nonpoint/

²³ United States Census Bureau, Table B25040, 2017: ACS 1-Year Estimates Detailed Tables

²⁴ See document titled “Residential Heating NEMO 2017 FINAL_4-2 update” located at: ftp://newftp.epa.gov/air/nei/2017/doc/supporting_data/nonpoint/

6.9.5 NATA Modeling

According to the 2014 NATA report, the total cancer risk determined for census tract ID 36029006800, representing Buffalo, NY, was 0.34 cancer cases per million residents. The six major drivers of this risk were Arsenic, Beryllium, Cadmium, Hexavalent Chromium, Formaldehyde, and Nickel. Additionally, a full change from traditional residential fuel oil to biomass-based diesel represents a 17% reduction in volatile organic compound emissions and an 86% reduction in particulate metal emissions.²⁵ In total, a full switch from residential fuel oil to biomass diesel represents a baseline risk change from 0.34 cases per million residents to 0.09 cases per million residents, a 0.25 case per million resident reduction. Table 6-19 below shows the detailed NATA cancer risk values and associated reductions.

Table 6-19. Buffalo NATA Risk Reduction for Census Tract 36029006800

Pollutant Name	NATA Cancer Risk (1 in 10 ⁶)	% Reduction	Reduced Risk (1 in 10 ⁶)
1,3-Butadiene	2.0E-03	17%	1.7E-03
Acetaldehyde	4.7E-03	17%	3.9E-03
Arsenic Compounds(Inorganic Including Arsine)	9.0E-02	86%	1.3E-02
Benzene	8.0E-03	17%	6.6E-03
Benzyl Chloride	2.1E-04	17%	1.8E-04
Beryllium Compounds	2.2E-02	86%	3.1E-03
Bromoform	2.7E-07	17%	2.2E-07
Chromium VI (Hexavalent)	3.6E-02	86%	5.1E-03
Cadmium Compounds	3.3E-02	86%	4.7E-03
Ethylbenzene	4.6E-05	17%	3.9E-05
Ethylene Dibromide (Dibromoethane)	1.2E-05	17%	1.0E-05
Formaldehyde	3.3E-02	17%	2.7E-02
Methyl Tert-Butyl Ether	5.6E-08	17%	4.7E-08
Nickel Compounds	1.0E-01	86%	1.4E-02
Naphthalene	1.3E-02	17%	1.0E-02
PAHs	3.6E-04	17%	3.0E-04
Total	0.34	74%	0.09

6.9.6 Census-Specific Modeling

Utilizing the emission rates outlined in Section 6.9.3, a modeling analysis specific to the Buffalo, NY census tract (census tract ID 36029006800) was completed. The three major drivers of this risk analysis were Arsenic, Cadmium, and Hexavalent Chromium. In this scenario, the baseline risk levels were substantially higher at 5.95 cases per million residents. This difference is accounted for both by a larger representation of metal emissions from a more refined emissions inventory, and higher pollutant toxicity values derived by OEHHA as used in the HARP analysis. With the associated volatile and metal risk reduction from a full switch to biomass-based diesel, the baseline risk is reduced to an average total risk of 0.86 cases per million

²⁵ Biodiesel emission reduction factors from "Proposed Emission Reductions for Public Health Benefits from Biodiesel Study;" email from Floyd Vergara, CFAA, to Jim Lyons, Trinity; August 4, 2020.

residents, representing a reduction of 5.1 cases per million residents. The table below shows the detailed modeled cancer risk values and associated reductions.

Table 6-20. Buffalo Census-Specific Modeling

Name	AERMOD Conc. (µg/m3)	HARP Risk	Reduction	Reduced Risk
Acetaldehyde	2.36E-04	2.1E-03	17%	1.8E-03
Arsenic	2.70E-05	3.6	86%	5.1E-01
B[a]anthracene	1.93E-07	1.0E-03	86%	1.4E-04
Benzene	1.03E-05	9.2E-04	17%	7.6E-04
Beryllium	2.02E-05	0.2	86%	2.1E-02
Cadmium	2.02E-05	0.3	86%	3.8E-02
Cr(VI)	3.64E-06	1.8	86%	0.3
Chrysene	1.14E-07	6.1E-05	86%	8.5E-06
D[a,h]anthracene	8.01E-08	1.6E-03	86%	2.3E-04
Formaldehyde	1.58E-03	3.0E-02	17%	2.5E-02
In[1,2,3-cd]pyr	1.03E-07	5.5E-04	86%	7.7E-05
Lead	6.07E-05	4.4E-02	86%	6.1E-03
Naphthalene	5.41E-05	5.8E-03	86%	8.1E-04
Nickel	2.02E-05	1.6E-02	86%	2.3E-03
Total	N/A	5.95	85.4%	0.86

6.9.7 Buffalo, NY Health Risk Summary

Given the conservative nature of the OEHHA health risk values utilized in the HARP software, it is expected that the actual cancer risk associated with residential heating oil usage in Buffalo, NY (census tract ID 36029006800) lies somewhere between 0.34 and 5.95 excess cancer cases per million residents over a 70-year timeline. Utilizing 17% and 86% reductions for volatile and metal toxics emissions, respectively, assuming a full transition from residential distillate heating oil to biomass-based diesel, that baseline risk is reduced to a value between 0.09 and 0.86 excess cancer cases per million residents.

6.9.8 Extrapolation of Risk Results to Other Similar Housing Areas

Risk results from Buffalo can be extrapolated to other areas with similar housing densities utilizing a **“density factor”**, which is the ratio of households utilizing fuel oils (23 for Census Tract 68) divided by the area (865,763 square meters for Census Tract 68). This data can be used to extrapolate the Census Tract 68 risk results to other areas based on the ratio of the Census Tract 68 **“density factor”** and associated risk (5.95 in one million) to other census tract **“density factors”** as follows, where Census Tract is abbreviated as CT:

Equation 1. Census Tract Density Factor

$$Density\ Factor_{CT\ x} = \frac{Homes\ Using\ Heating\ Oil_{CT\ x}}{Area\ (m^2)_{CT\ x}} / \frac{Homes\ Using\ Heating\ Oil_{CT68}}{Area\ (m^2)_{CT68}}$$

Equation 2. Unit Risk Factor

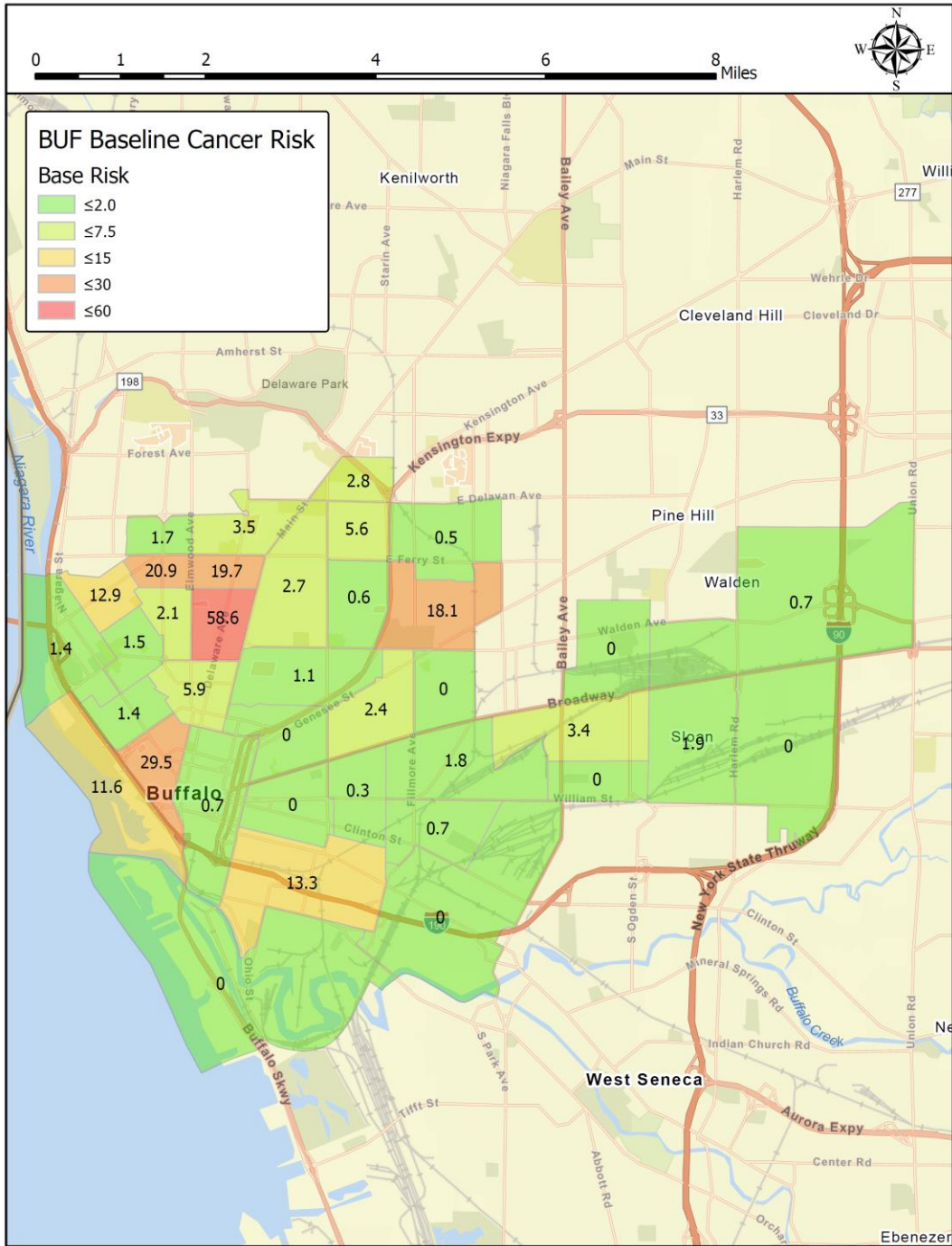
$$\text{Unit Risk} \left(\frac{\text{Risk}}{\text{Homes Using Heating Oil}} \right) = \frac{\text{Risk}_{CT68}}{\text{Homes Using Heating Oil}_{CT68}}$$

Equation 3. Extrapolated Census Tract Risk

$$\text{Risk}_{CT X} = \text{Homes Using Heating Oil}_{CT X} * \text{Unit Risk Factor} * \text{Density Factor}_{CT X}$$

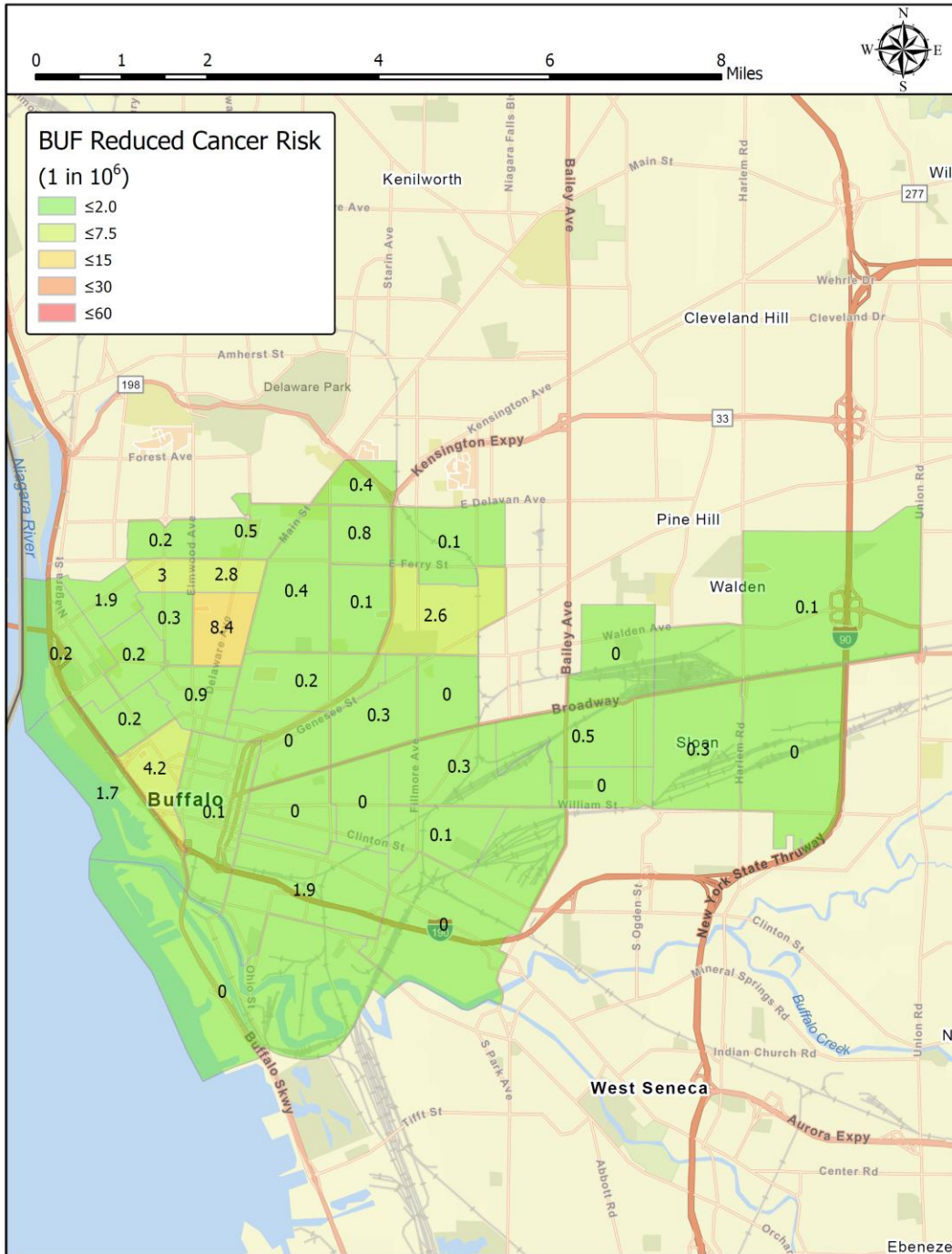
In this case, Census Tract 68 has 23 houses burning distillate oil and an area of 865,763 square meters, so a density factor of 2.66E-05 fuel oil houses per meter squared. This factor, combined with the Census Tract 68 unit risk factor of 0.2586 (5.95 per million cases/23 houses), can then be multiplied by the number of residences using heating oil in any other nearby census tract to get an estimated risk due to distillate oil combustion in that tract. The figure below shows the calculated risk per million residents due to distillate oil combustion in the vicinity (~8-mile diameter area) of Buffalo, NY near Census Tract 68.

Figure 6-83. Buffalo Cancer Risk Due to Home Heating Oil Combustion



The figure below shows the reduction in risk due to the conversion to biomass-based diesel fuel for the same 8-mile diameter area surrounding Buffalo Census Tract 68.

Figure 6-84. Buffalo Reduced Cancer Risk Due to Biodiesel Heating Oil Combustion



The reduction in “cancer burden”, which is just each respective census tract risk multiplied by the census tract population divided by 1 million, can be determined from this same data. The maximum baseline cancer burden is 0.17 for census tract 36029006702, compared to Census Tract 68 cancer burden of 0.02, indicating that this census tract could expect <1 person to develop cancer over a 70-year lifetime due to exposure to distillate oil combustion within that census tract alone. The total cancer burden for all of the census tracts in the selected 8-mile radius is 0.64, with an expected reduction in cancer burden to a value of approximately 0.09 with the use of biomass-based diesel fuel for home heating oil combustion.

6.9.9 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-21 below.

Table 6-21. Buffalo Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	29.7	\$910,824
Asthma Symptoms - Albuterol use	2,879	\$995
ER visits - All Cardiac Outcomes	2.8	\$3,283
ER visits – Respiratory	5.0	\$4,347
HA – All – Respiratory	0.8	\$13,149
HA – Alzheimer's Disease	2.7	\$32,701
HA – Cardio Cerebro- and Peripheral Vascular Disease	1.4	\$21,944
HA – Parkinson's Disease	0.8	\$10,575
HA – Respiratory-2	0.2	\$0
HA – Respiratory-2 HA – All Respiratory	0.9	\$0
Incidence – Asthma	21.6	\$964,107
Incidence – Hay Fever/Rhinitis	139.7	\$83,816
Incidence – Lung Cancer	1.4	\$18,054
Incidence – Out of Hospital Cardiac Arrest	0.2	\$5,691
Incidence – Stroke	0.6	\$20,853
Minor Restricted Activity Days	7,206	\$501,420
Mortality – All Cause	12.6	\$98,265,408
Work Loss Days	1,214	\$216,561
Total		\$101,073,727

6.10 Port of Elizabeth, New York

6.10.1 NATA Health Risks

The subsections below review the NATA data available for the Port of Elizabeth, NY (Port of Elizabeth) community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-85 and Figure 6-86 show the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

Figure 6-87 and Figure 6-88 show those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-89 and Figure 6-90 show the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Port of Elizabeth community.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the Port of Elizabeth community.

6.10.1.1 NATA Risk Data

Figure 6-85. Port of Elizabeth North Baseline NATA Total Cancer Risks

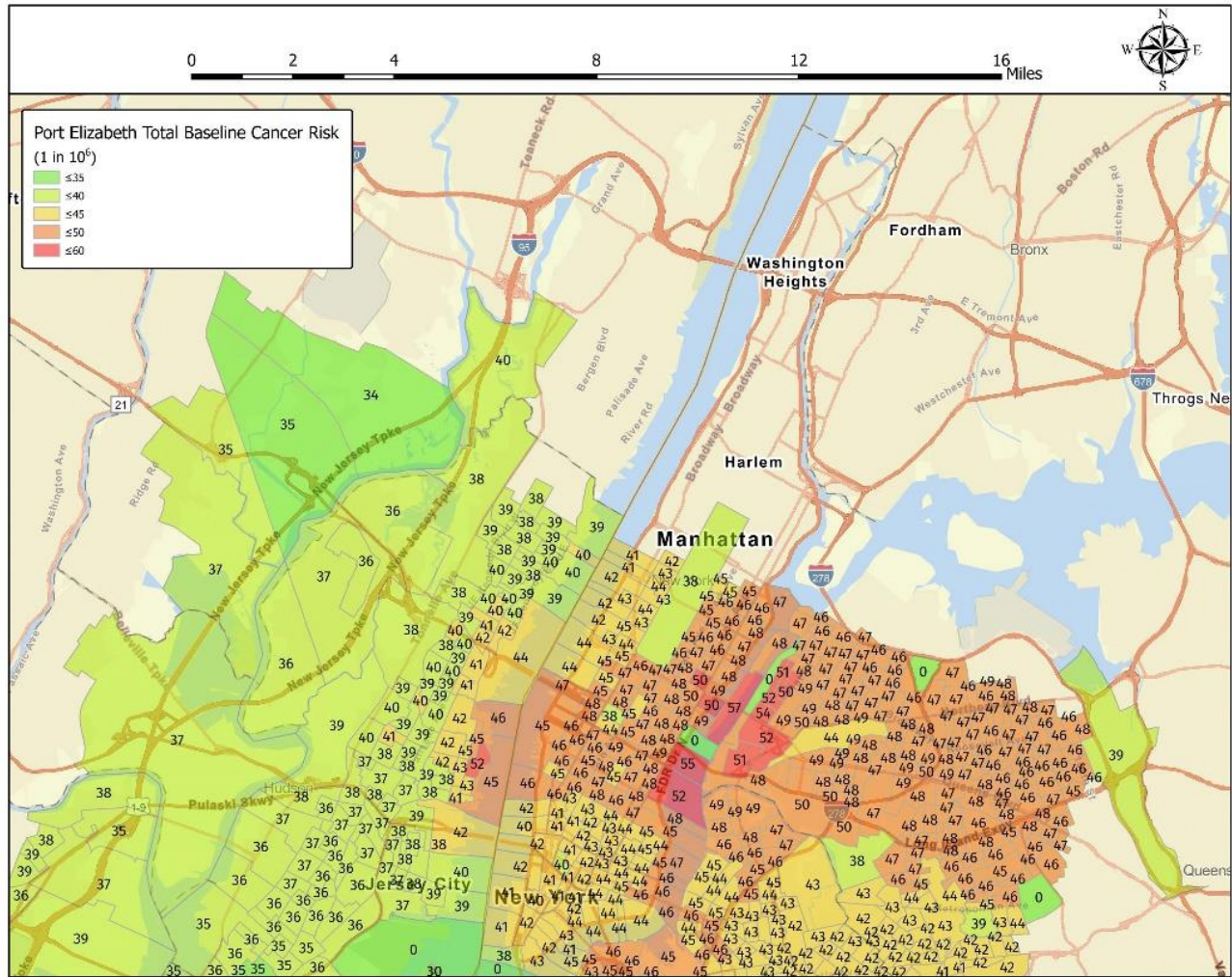
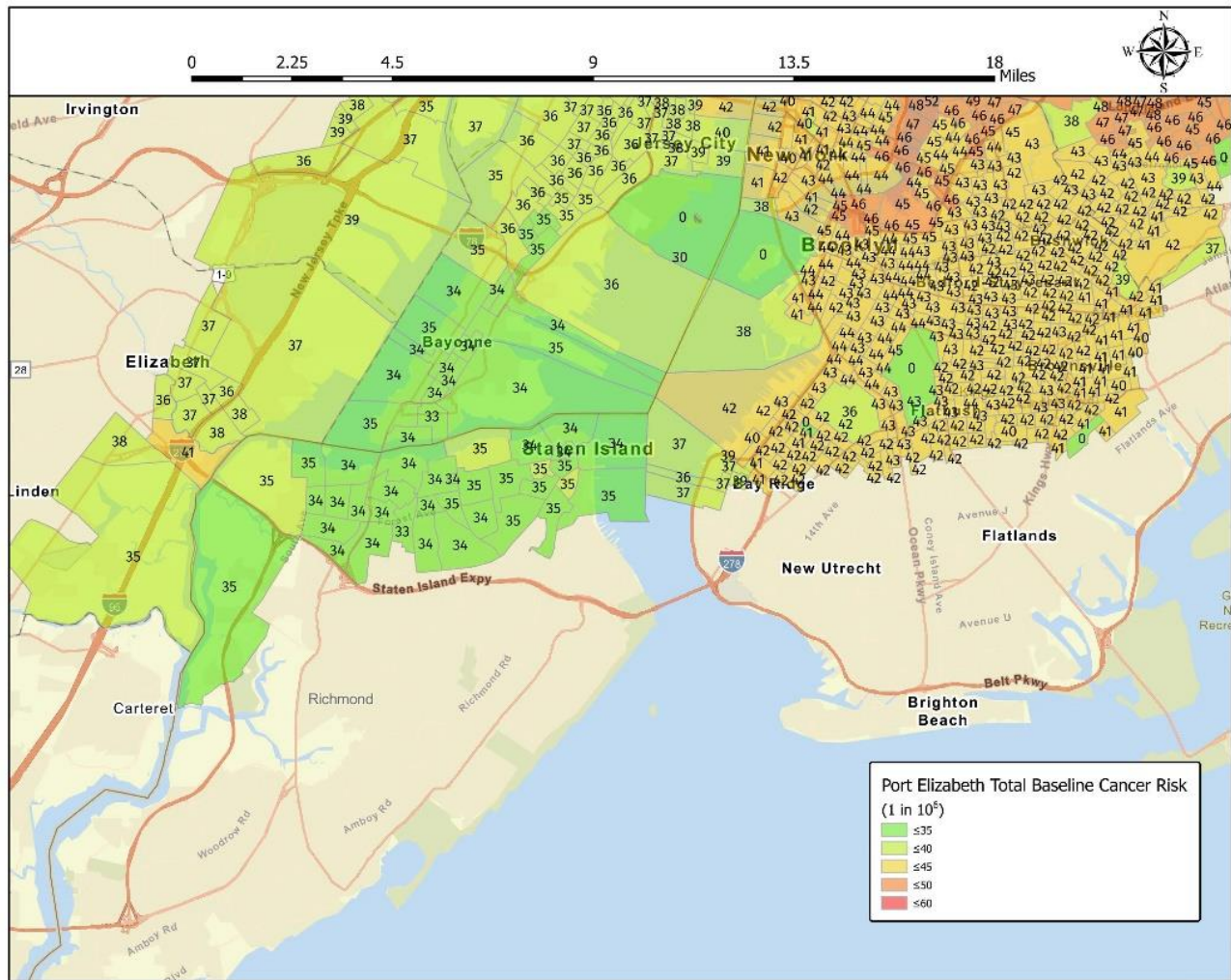


Figure 6-86. Port of Elizabeth South Baseline NATA Total Cancer Risks



According to the NATA, the maximum baseline cancer risk in the Port of Elizabeth community is 57 cancer cases per million residents for census tract 36061023801, with a population of 9,723 residents. When accounting for all of the communities assessed, the total cancer burden for the Port of Elizabeth community is 164 cancer cases expected over a 70-year timeline among a total community population of 3,823,511.

Figure 6-87. Port of Elizabeth North Baseline NATA DPM Cancer Risks

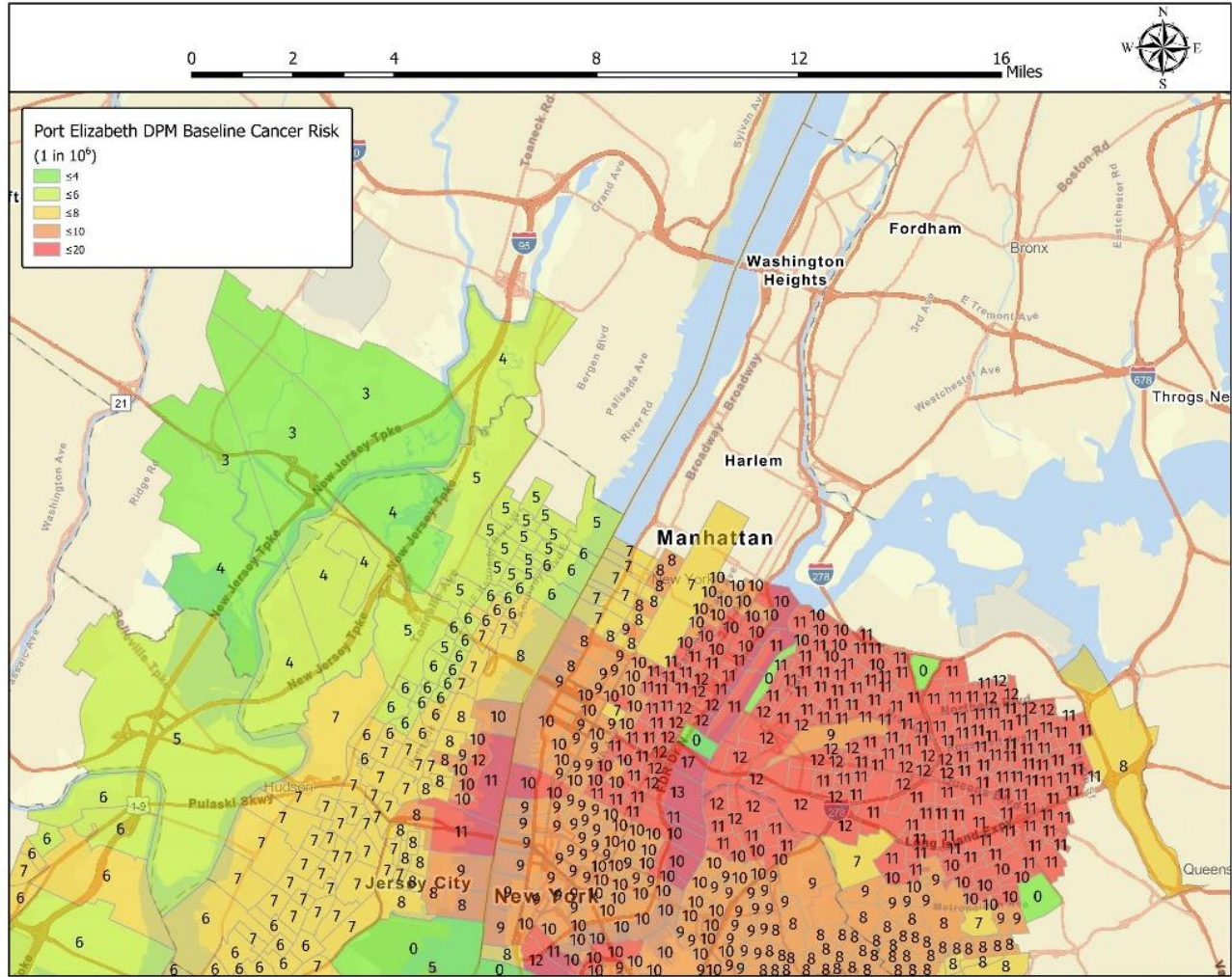
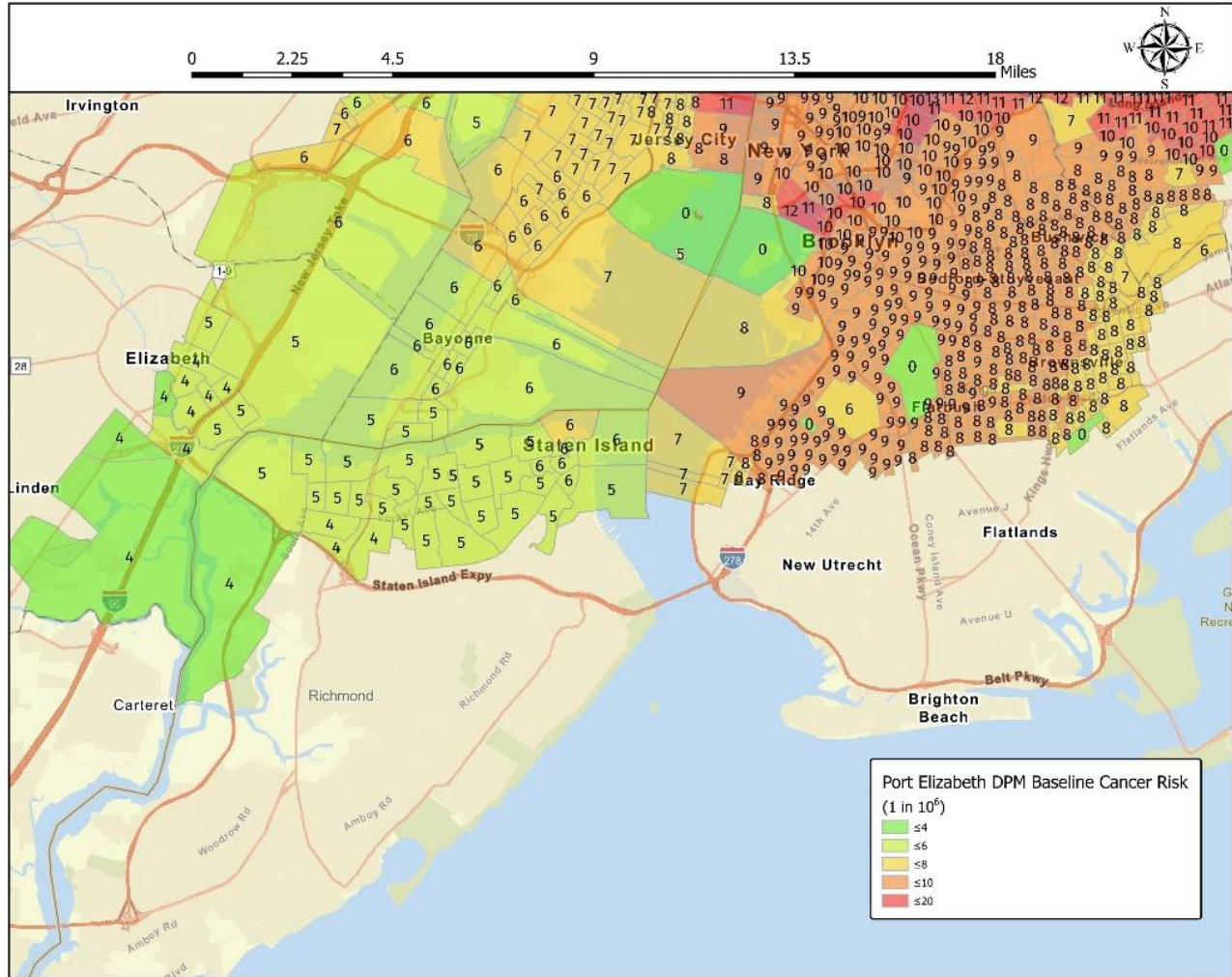


Figure 6-88. Port of Elizabeth South Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the Port of Elizabeth community is 17 cancer cases per million residents for census tract 36061008601, with a population of 2,618 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Port of Elizabeth community is 33 cancer cases expected over a 70-year timeline among a total community population of 3,823,511.

Figure 6-89. Port of Elizabeth North Reduced NATA DPM Cancer Risks

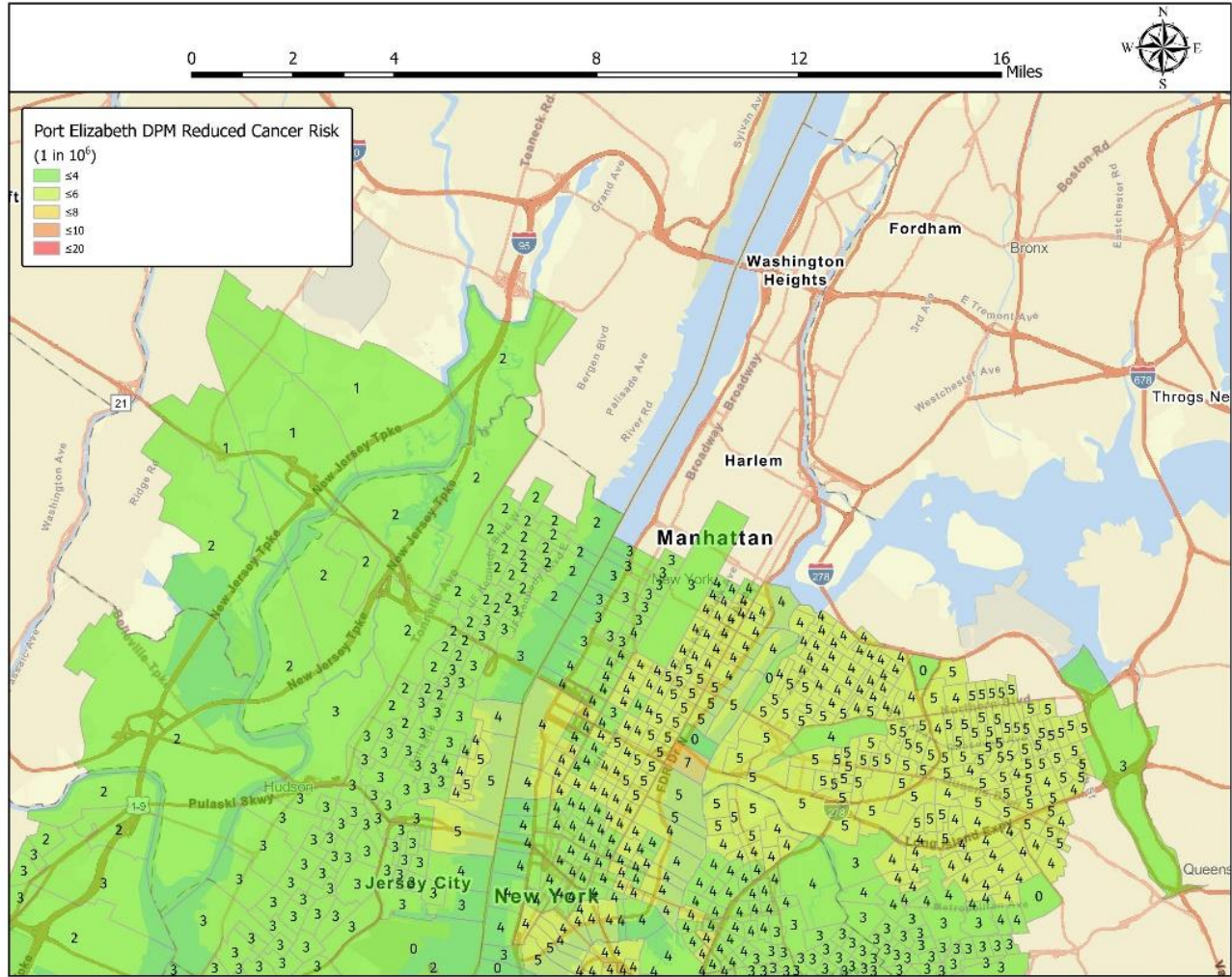
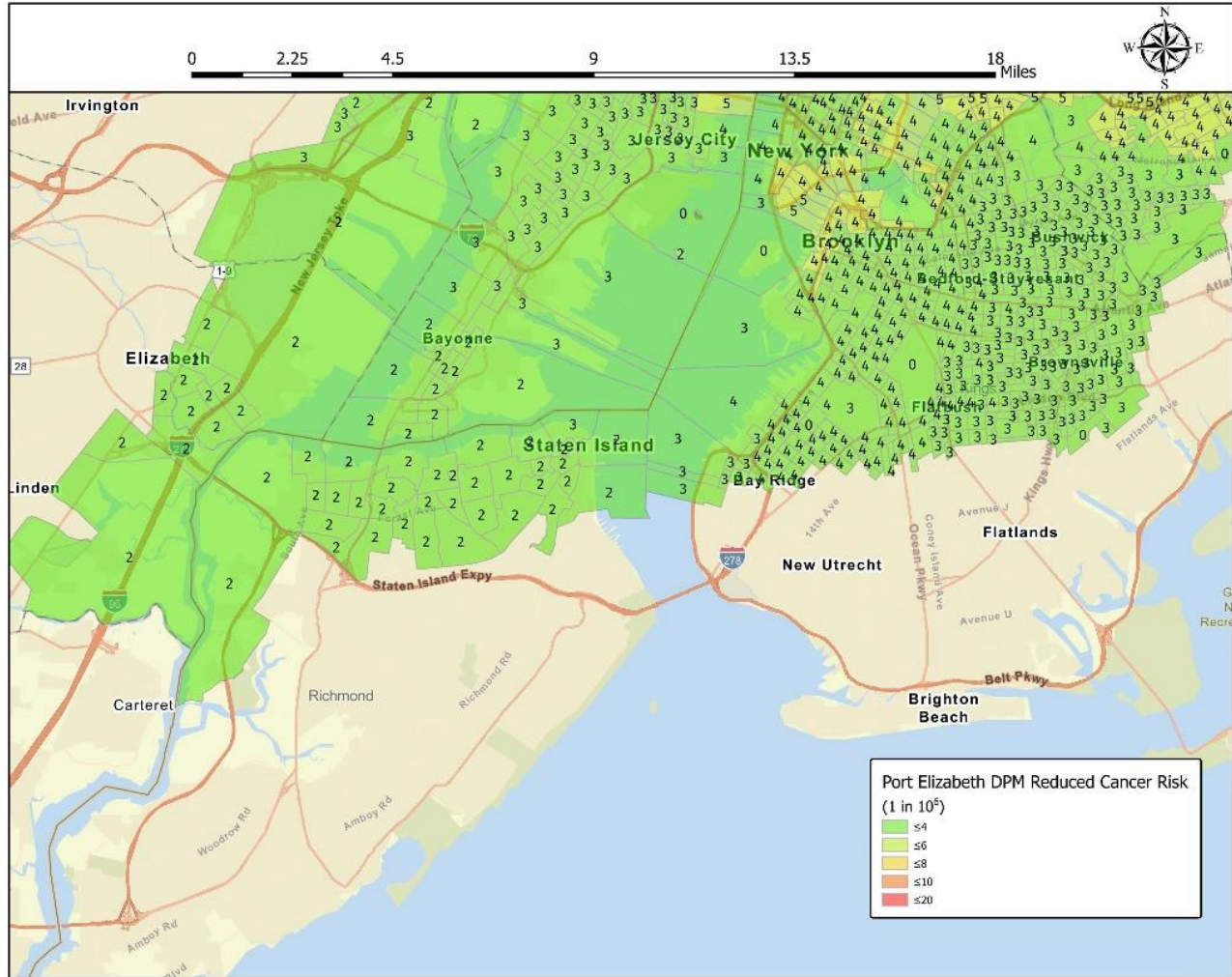


Figure 6-90. Port of Elizabeth South Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Port of Elizabeth community becomes 7 cancer cases per million residents for census tract 36061008601, with a population of 2,618 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Port of Elizabeth community becomes 14 cancer cases expected over a 70-year timeline among a total community population of 3,823,511.

6.10.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB’s HARP program with OEHHA cancer unit risk values.** The data is outlined in the following order:

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program.**

Figure 6-91 and Figure 6-92 show the baseline DPM concentrations provided by the NATA.

Figure 6-93 and Figure 6-94 show the baseline DPM-specific cancer risks as determined using the NATA **concentration values and CARB's HARP program.**

Figure 6-95 and Figure 6-96 show the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Port of Elizabeth community.

Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the Port of Elizabeth community.

Figure 6-91. Port of Elizabeth North Baseline NATA DPM Concentrations

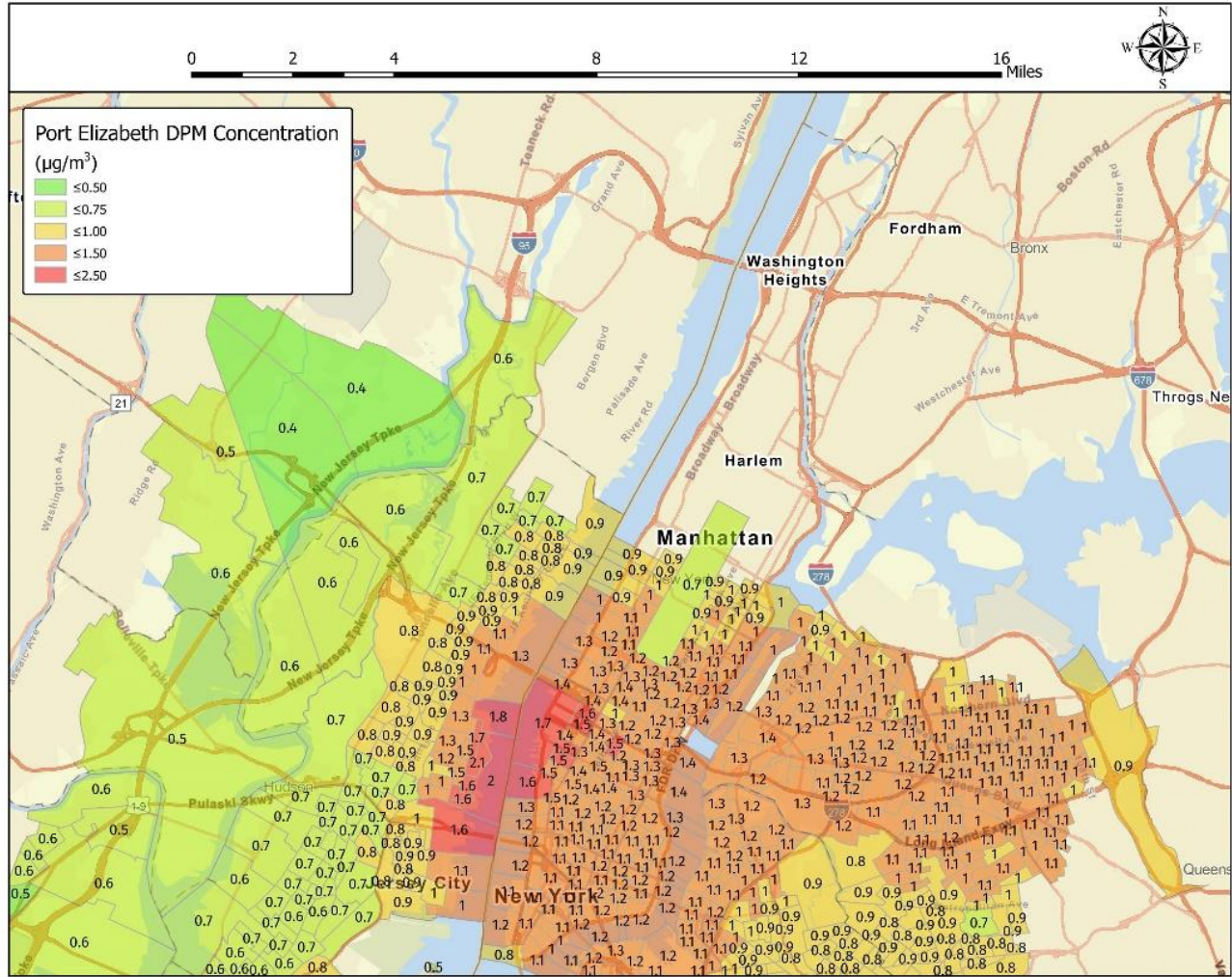
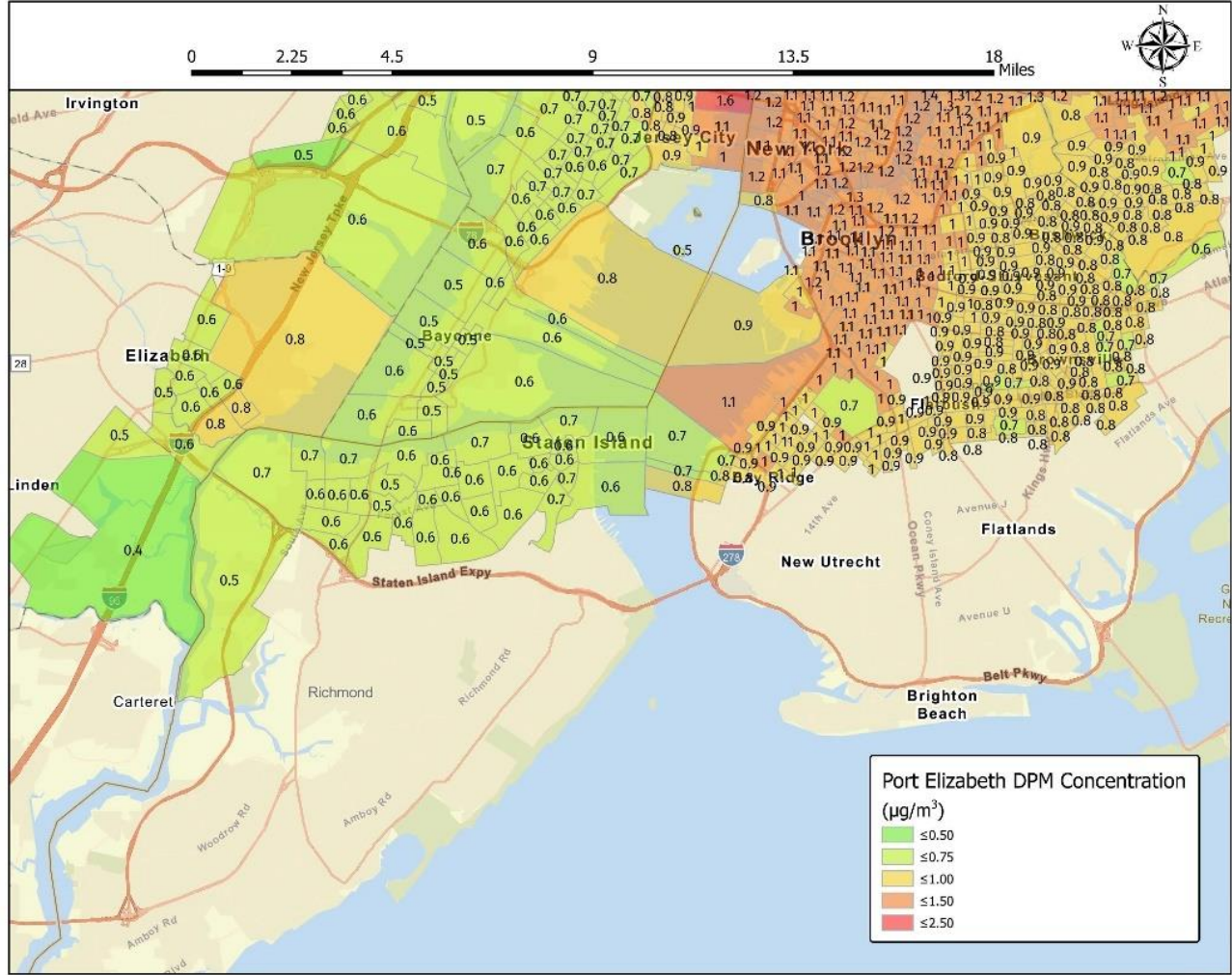


Figure 6-92. Port of Elizabeth South Baseline NATA DPM Concentrations



According to the NATA, the maximum baseline DPM concentration in the Port of Elizabeth community is 2.12 µg/m³ for census tract 34017018701, with a population of 2,936 residents. The average DPM concentration of the Port of Elizabeth community is 0.95 µg/m³.

Figure 6-93. Port of Elizabeth North Baseline NATA/HARP DPM Hybrid Risks

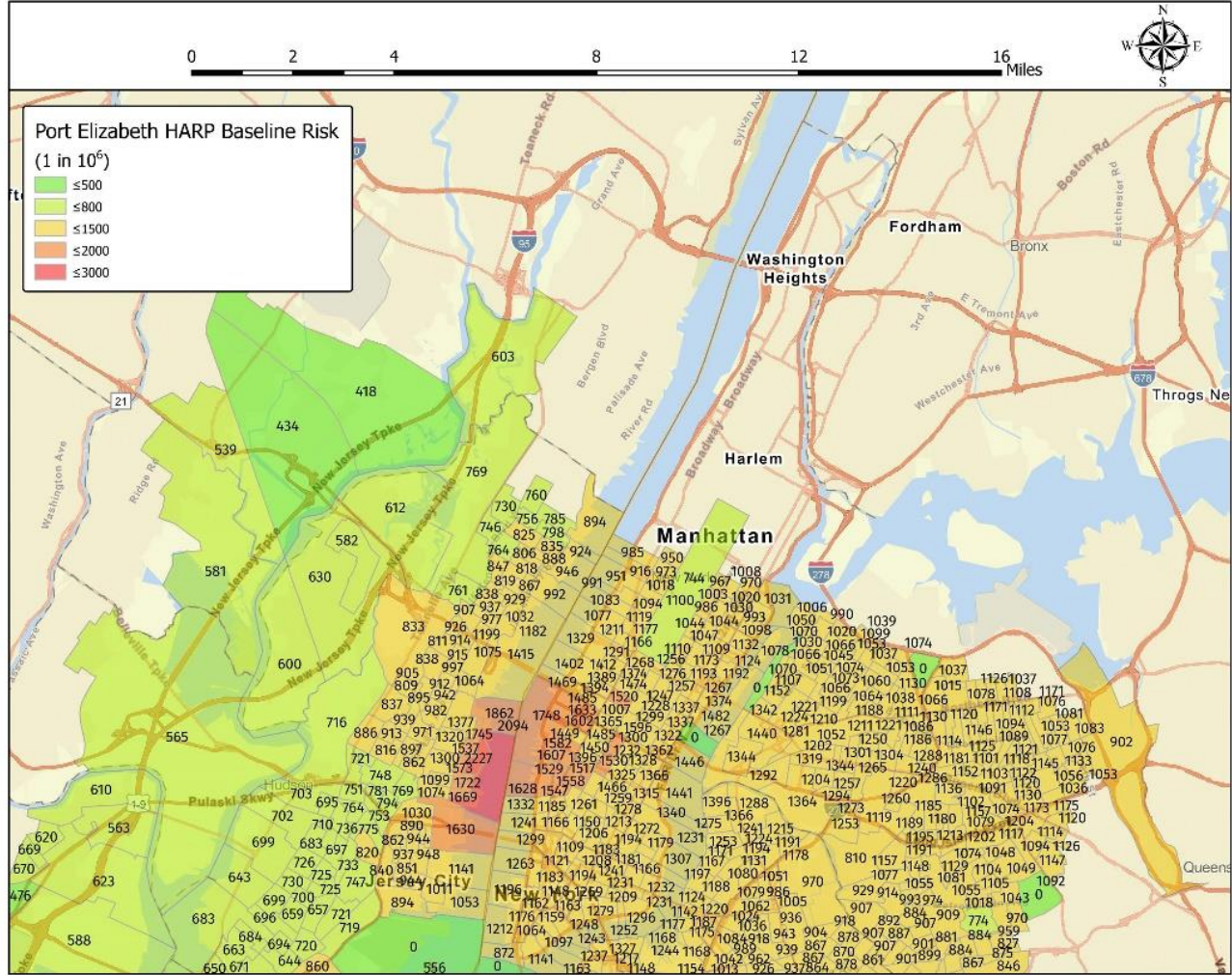
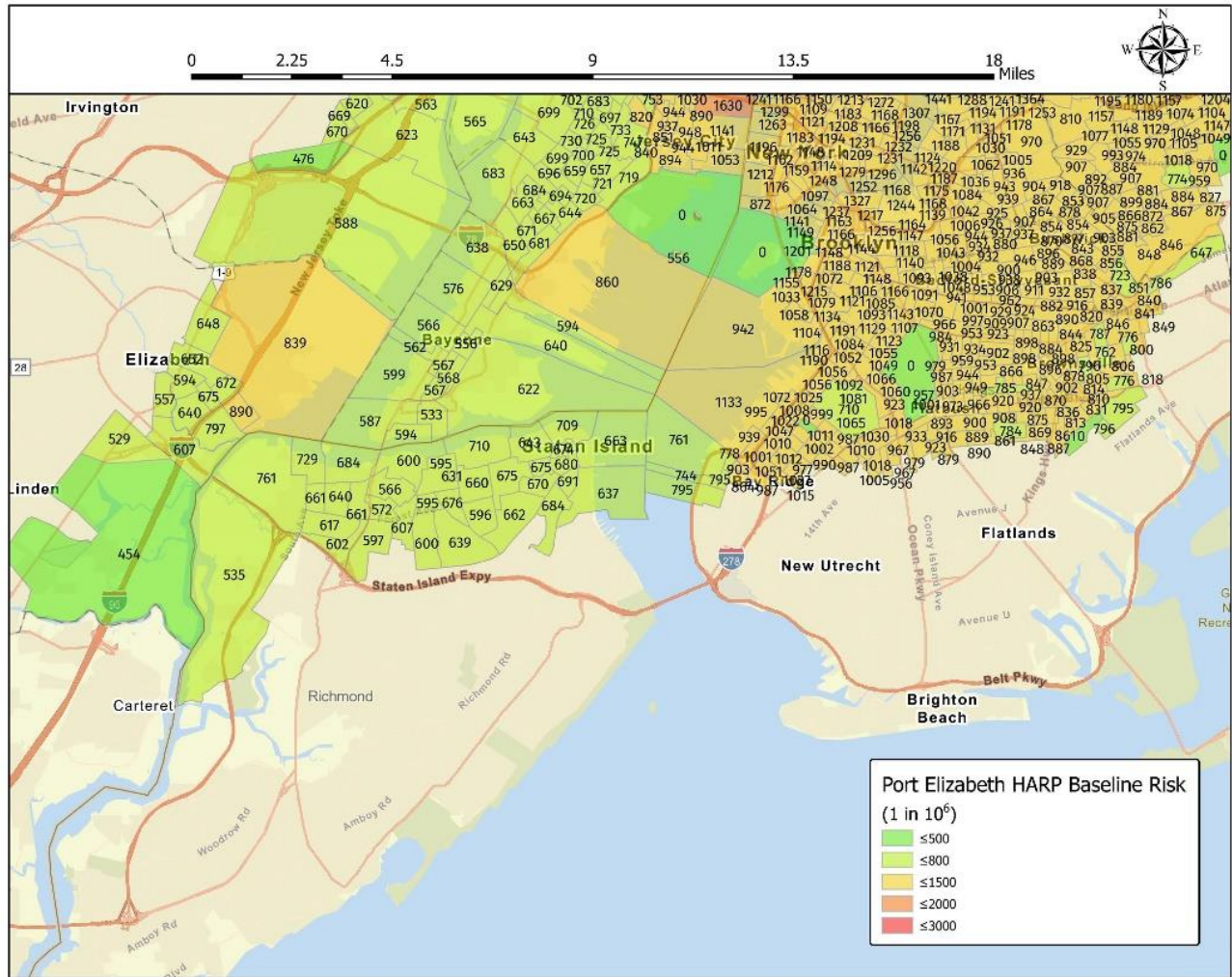


Figure 6-94. Port of Elizabeth South Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the Port of Elizabeth community is 2,227 cancer cases per million residents for census tract 34017018701, with a population of 2,936 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Port of Elizabeth community is 3,911 cancer cases expected over a 70-year timeline among a total community population of 3,823,511.

Figure 6-95. Port of Elizabeth North Reduced NATA/HARP DPM Hybrid Risks

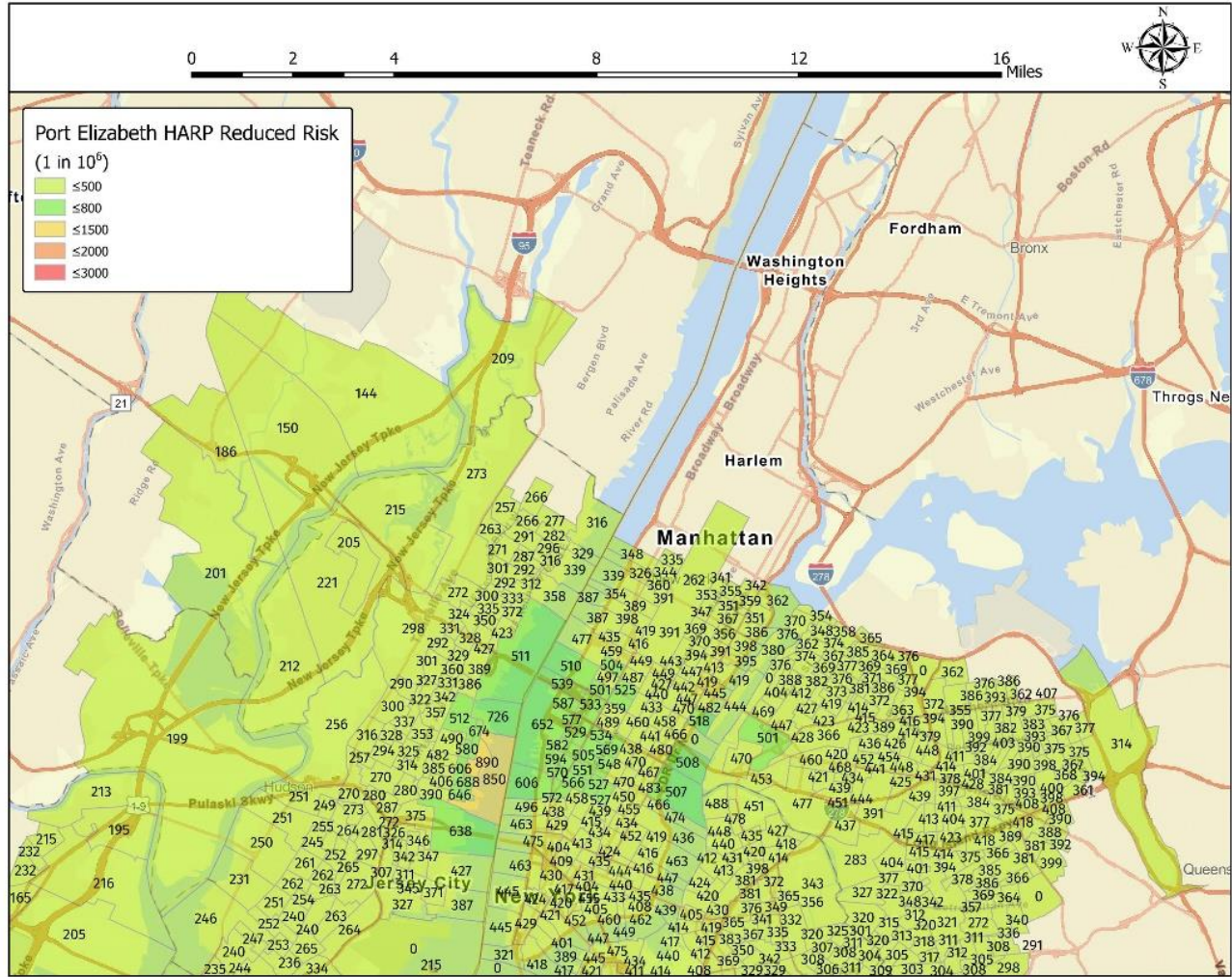
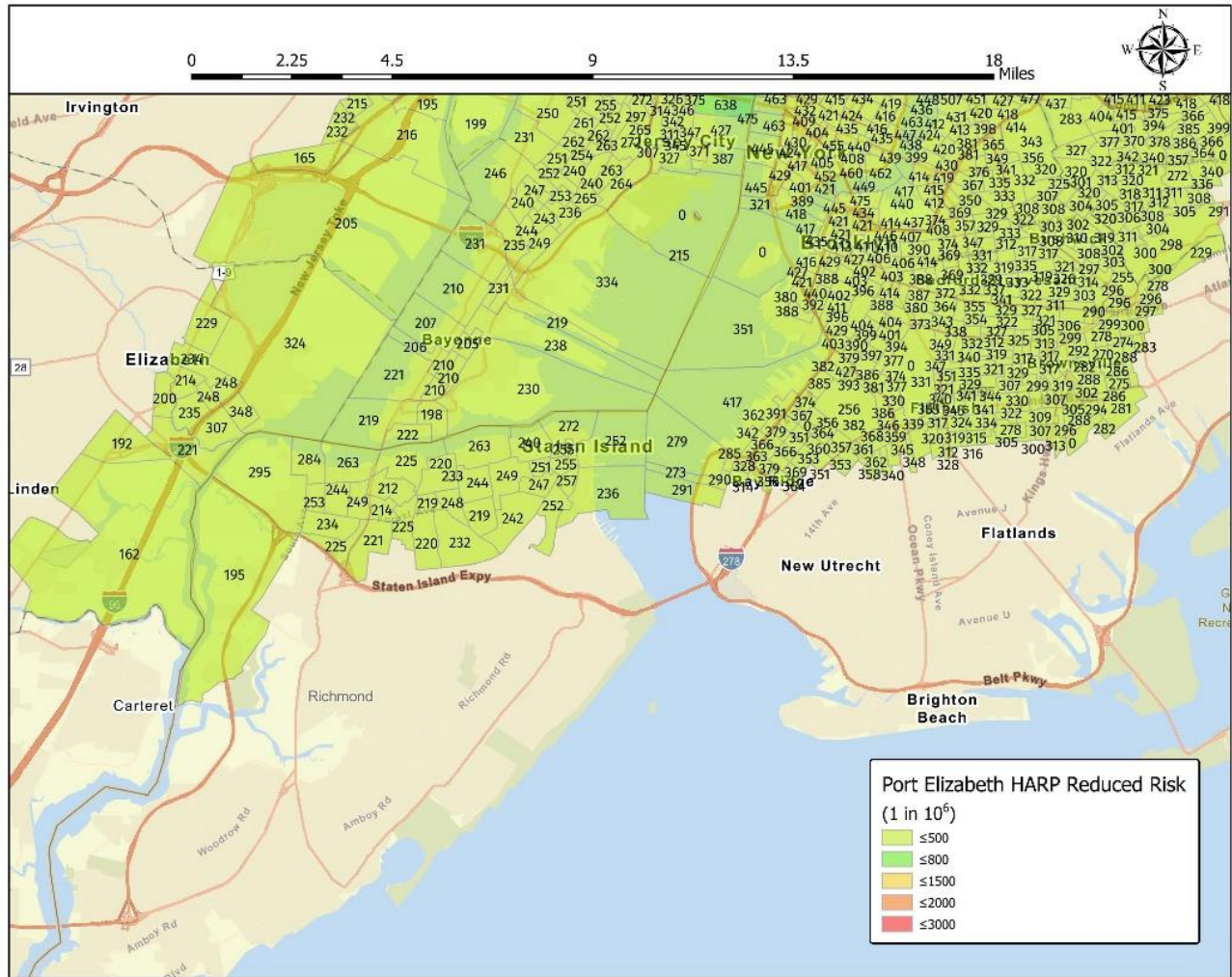


Figure 6-96. Port of Elizabeth South Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Port of Elizabeth community becomes 890 cancer cases per million residents for census tract 34017018701, with a population of 2,936 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Port of Elizabeth community becomes 1,394 cancer cases expected over a 70-year timeline among a total community population of 3,823,511.

6.10.2 Port of Elizabeth Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Port of Elizabeth. The following sources were utilized to generate the HRA.

- New Jersey Department of Transportation (NJDOT) – Traffic Counts (2019 Average Annual Daily Traffic)²⁶
- New York State Department of Transportation (NYSDOT) – Traffic Counts (2019 Average Annual Daily Traffic)²⁷

The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-22. Table 6-11.

Table 6-22. Port of Elizabeth Source Groups and Emission Rates

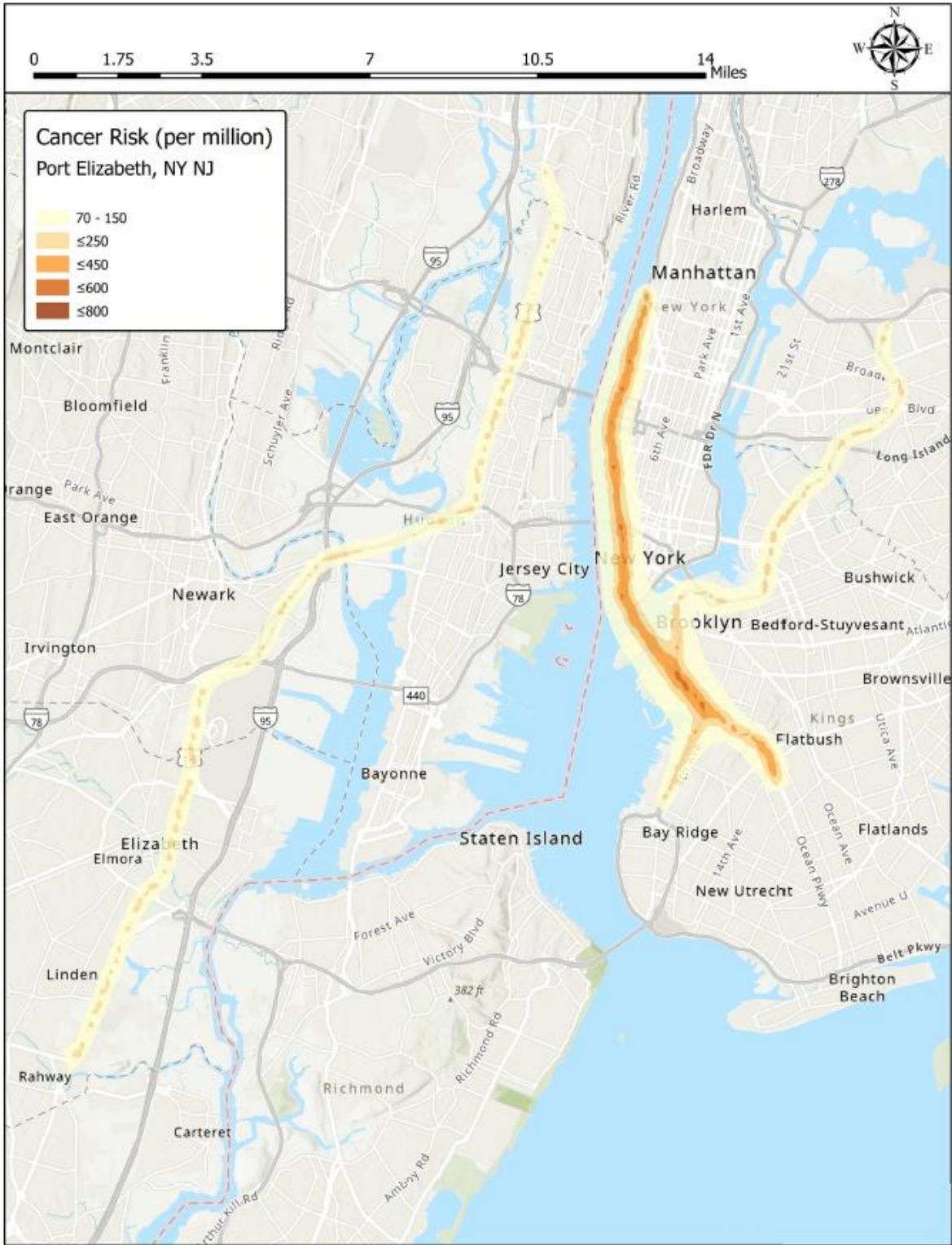
Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
9	State Route 9 – 64,934 AADT	14,967	59.7%
278	State Route 278 – 117,315 AADT	15,529	59.7%
27/478	State Route 27/478 – 78,981 AADT	8,998	59.7%
Williamsburg	Williamsburg – 80,478 AADT	1,428	59.7%

These sources were modeled with unit emission rates in AERMOD, and the Table 6-11. listed emission rates **were input into CARB's HARP software to determine cancer risks from** the DPM concentrations determined by AERMOD. While dispersion characteristics remained the same between baseline and reduced modeling scenarios, **emission rates were reduced according to the number of "old technology" engines combusting diesel, based on source type. The table above shows the Proportion of "Old Technology" Engine Emissions** where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

²⁶ <https://www.njtms.org/map/>

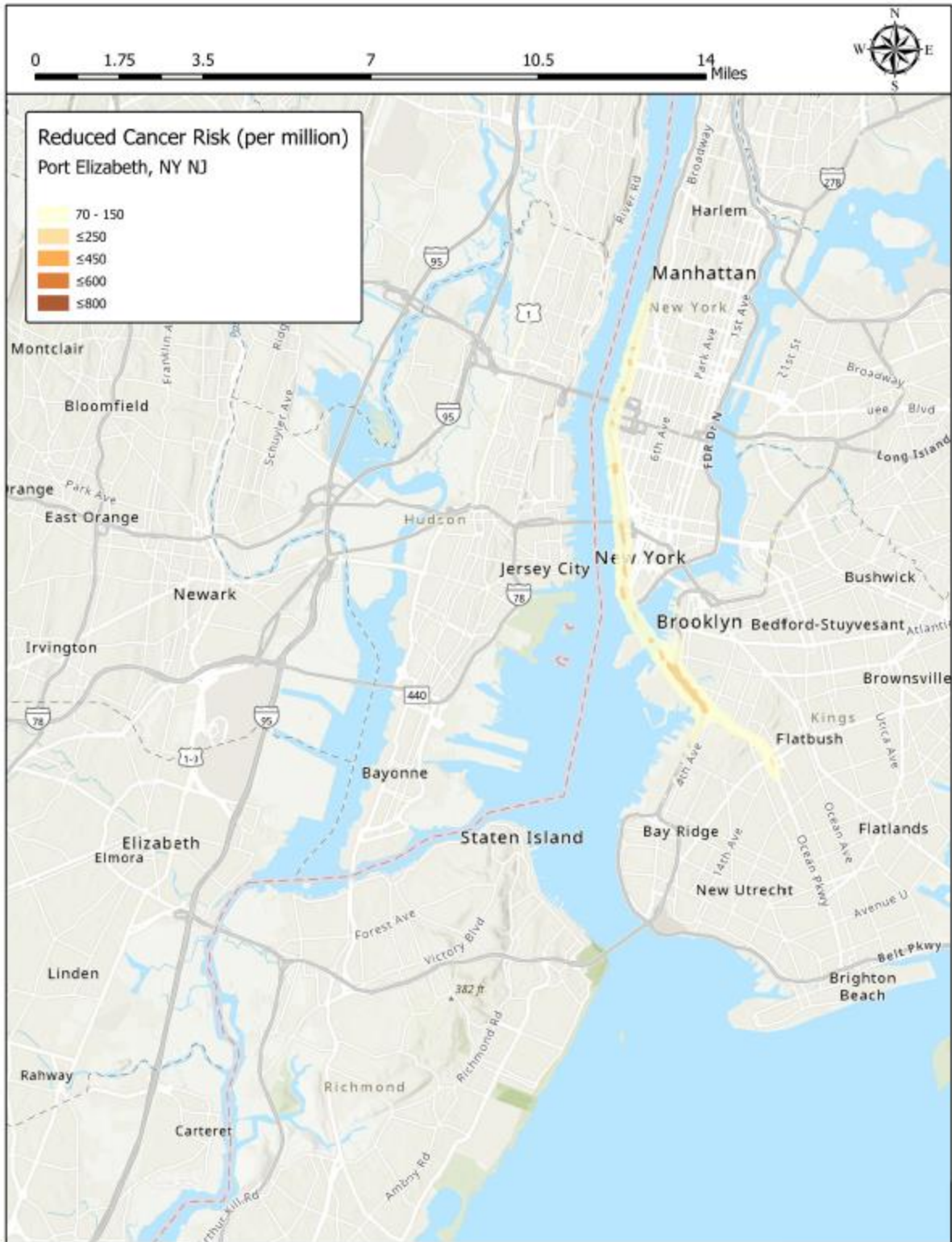
²⁷ <https://gisportalny.dot.ny.gov/portalny/apps/webappviewer/index.html?id=28537cbc8b5941e19cf8e959b16797b4>

Figure 6-97. Port of Elizabeth Baseline Health Risk Assessment Isoleths



The site-specific HRA shows that the point of maximum impact (PMI) is substantially lower than the NATA/HARP evaluation, with an impact of 768 cancer cases per million residents. This PMI does not occur at a residential receptor, though, and does not represent an actual risk to residences in the area. The MEIR occurs at 584,263.8 m E, and 4,503,419.3 m N (NAD 83, UTM Zone 17), with a baseline risk of 670 cancer cases per million residents. This MEIR is higher than the NATA/HARP hybrid risks evaluated for that census tract (36047005900) with a total risk of 380 in a million. This HRA does not capture all of the cancer-causing sources in the area but does demonstrate that NATA values are in-line with the site-specific demonstration with some extremely high local maxima due to local residences proximity to highways. Additional review of transportation sources is required to accurately assess local maxima of health risk impacts in the Port of Elizabeth community.

Figure 6-98. Port of Elizabeth Reduced Health Risk Assessment Isoleths



The reduced cancer risk PMI and MEIR are 309 and 270 in 1 million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 400 in 1 million at the MEIR.

6.10.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-23 below.

Table 6-23. Port of Elizabeth Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	480.2	\$15,115,170
Asthma Symptoms - Albuterol use	74,288	\$25,673
ER visits - All Cardiac Outcomes	56.8	\$65,966
ER visits – Respiratory	156.0	\$136,416
HA – All – Respiratory	24.1	\$359,484
HA – Alzheimer's Disease	96.6	\$1,191,555
HA – Cardio Cerebro- and Peripheral Vascular Disease	25.6	\$401,042
HA – Parkinson's Disease	15.1	\$195,970
HA – Respiratory-2	3.4	\$0
HA – Respiratory-2 HA – All Respiratory	27.5	\$0
Incidence – Asthma	574.6	\$25,658,684
Incidence – Hay Fever/Rhinitis	3,548	\$2,128,554
Incidence – Lung Cancer	29.0	\$364,106
Incidence – Out of Hospital Cardiac Arrest	3.5	\$124,068
Incidence – Stroke	11.4	\$388,636
Minor Restricted Activity Days	193,805	\$13,485,269
Mortality – All Cause	174.6	\$1,366,431,014
Work Loss Days	33,296	\$6,880,283
Total		\$1,432,951,890

6.11 Charlotte, North Carolina

6.11.1 NATA Health Risks

The subsections below review the NATA data available for the Charlotte, NC (Charlotte) community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-99 shows the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

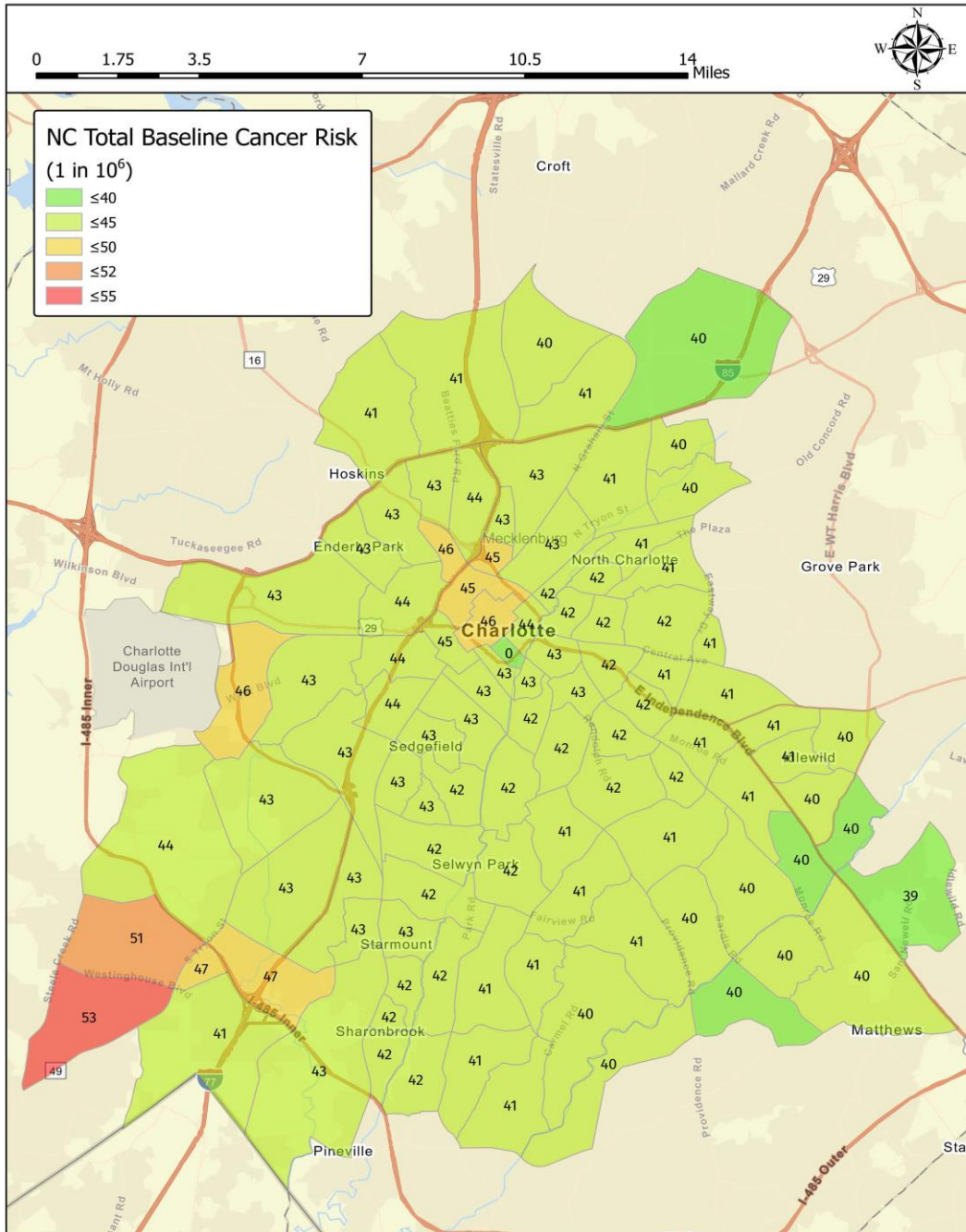
Figure 6-100 shows those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-101 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Charlotte community.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the Charlotte community.

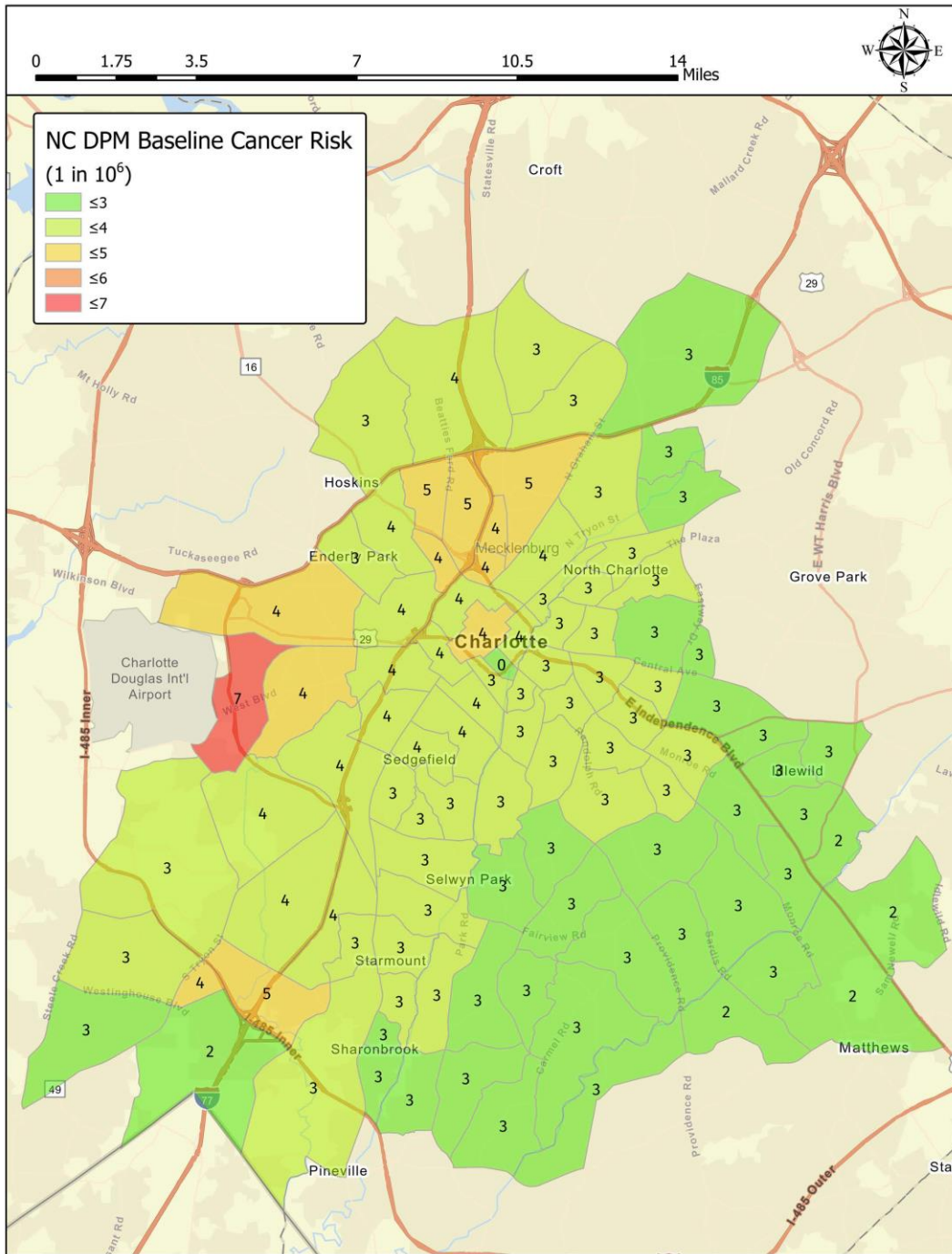
6.11.1.1 NATA Risk Data

Figure 6-99. Charlotte Baseline NATA Total Cancer Risks



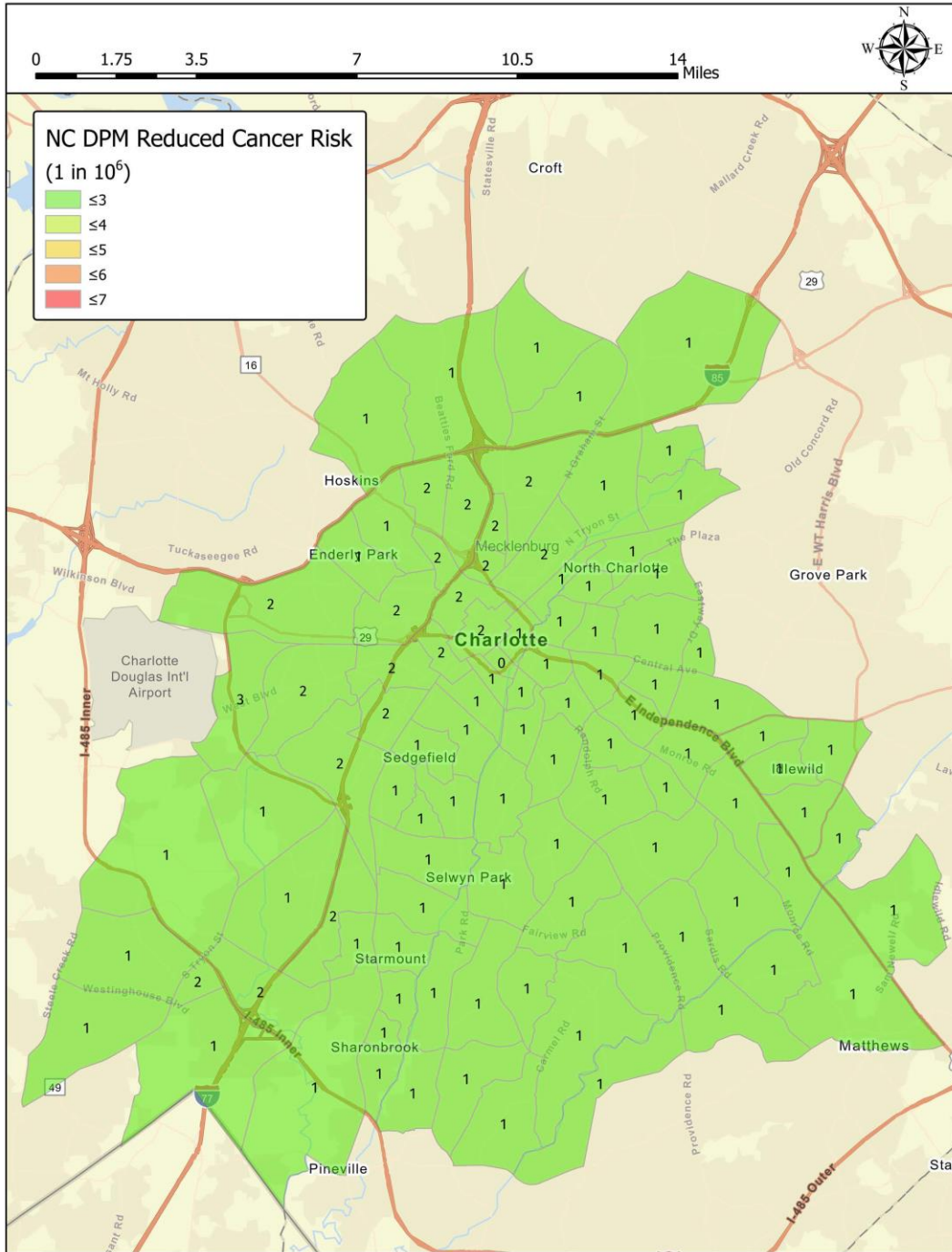
According to the NATA, the maximum baseline cancer risk in the Charlotte community is 52.80 cancer cases per million residents for census tract 37119005914, with a population of 5,563 residents. When accounting for all of the communities assessed, the total cancer burden for the Charlotte community is 15 cancer cases expected over a 70-year timeline among a total community population of 361,062.

Figure 6-100. Charlotte Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the Charlotte community is 7 cancer cases per million residents for census tract 37119003903, with a population of 2,541 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Charlotte community is 1 cancer case expected over a 70-year timeline among a total community population of 361,062.

Figure 6-101. Charlotte Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Charlotte community becomes 3 cancer cases per million residents for census tract 37119003903, with a population of 2,541 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Charlotte community becomes less than 1 cancer case expected over a 70-year timeline among a total community population of 361,062.

6.11.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB's HARP program with OEHHA** cancer unit risk values. The data is outlined in the following order:

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with baseline and reduced cancer risks using **CARB's HARP program**.

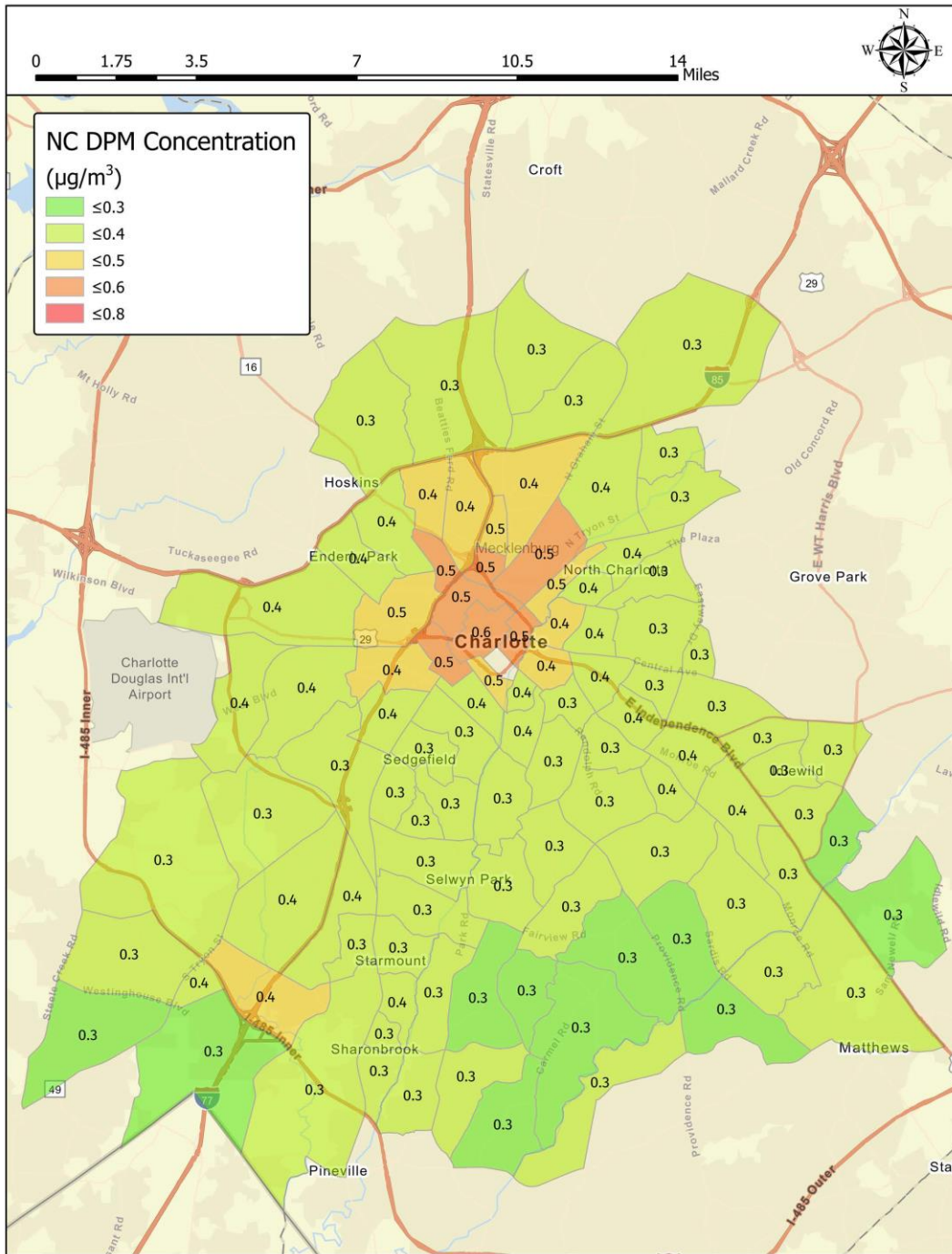
Figure 6-102 shows the baseline DPM concentrations provided by the NATA.

Figure 6-103 shows the baseline DPM-specific cancer risks as determined using the NATA concentration **values and CARB's HARP program**.

Figure 6-104 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Charlotte community.

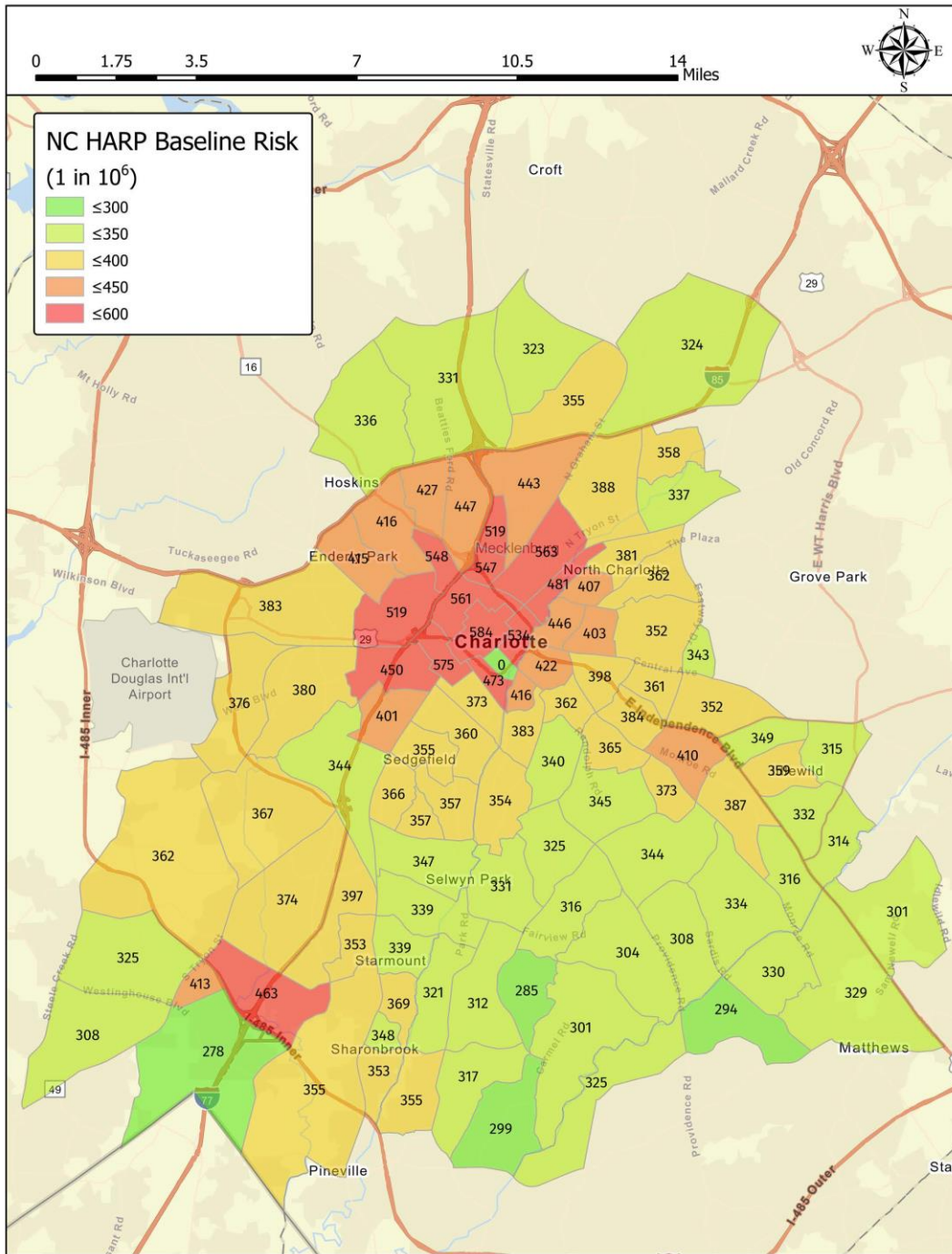
Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the Charlotte community.

Figure 6-102. Charlotte Baseline NATA DPM Concentrations



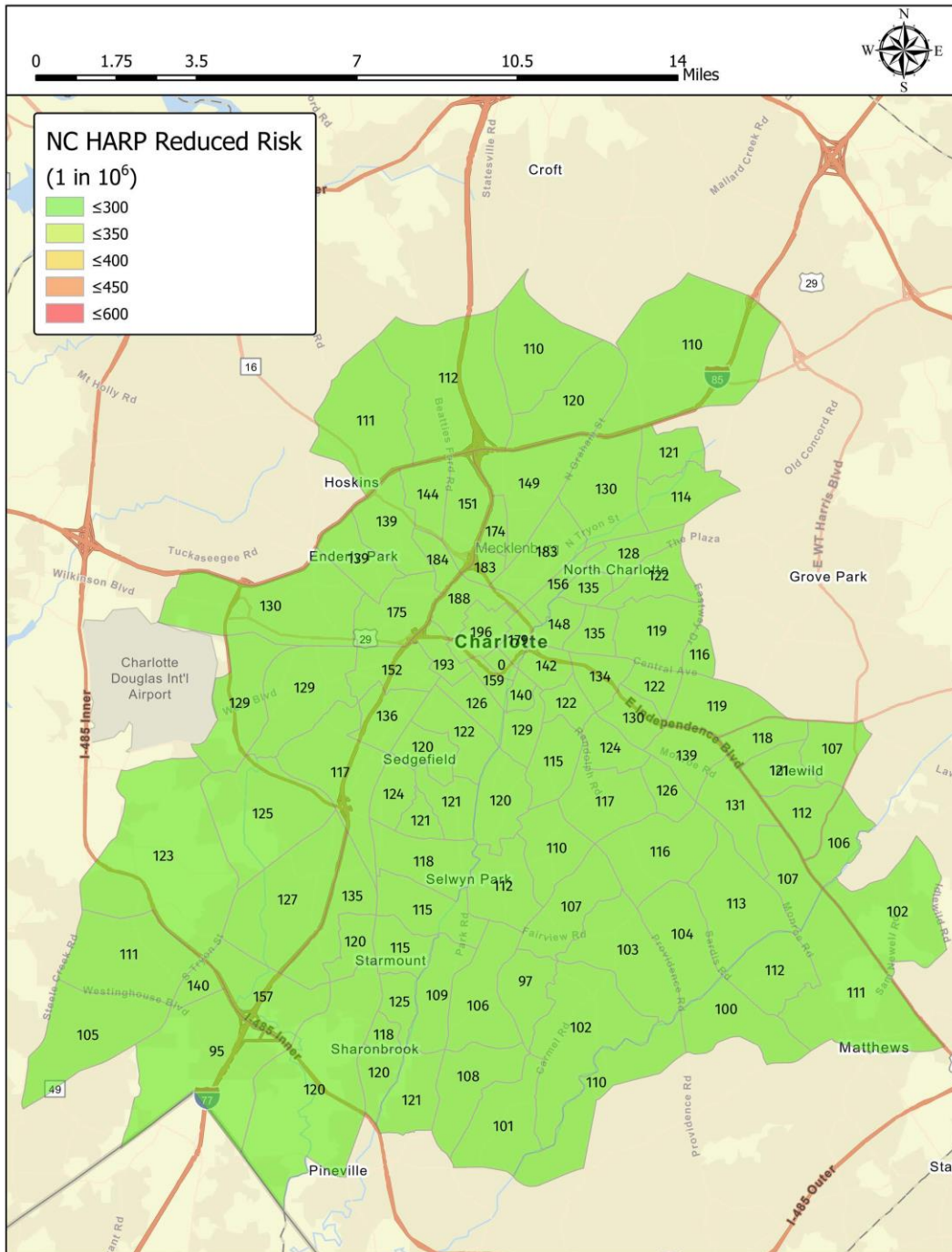
According to the NATA, the maximum baseline DPM concentration in the Charlotte community is 0.56 $\mu\text{g}/\text{m}^3$ for census tract 37119000100, with a population of 3,425 residents. The average DPM concentration of the Charlotte community is 0.36 $\mu\text{g}/\text{m}^3$.

Figure 6-103. Charlotte Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the Charlotte community is 584 cancer cases per million residents for census tract 37119000100, with a population of 3,425 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Charlotte community is 131 cancer cases expected over a 70-year timeline among a total community population of 361,062.

Figure 6-104. Charlotte Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Charlotte community becomes 196 cancer cases per million residents for census tract 37119000100, with a population of 3,425 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Charlotte community becomes 44 cancer case expected over a 70-year timeline among a total community population of 361,062.

6.11.2 Charlotte Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Charlotte. The following sources were utilized to generate the HRA.

- North Carolina Department of Transportation (NCDOT) – Traffic Counts (2019 Average Annual Daily Traffic)²⁸

The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-11.

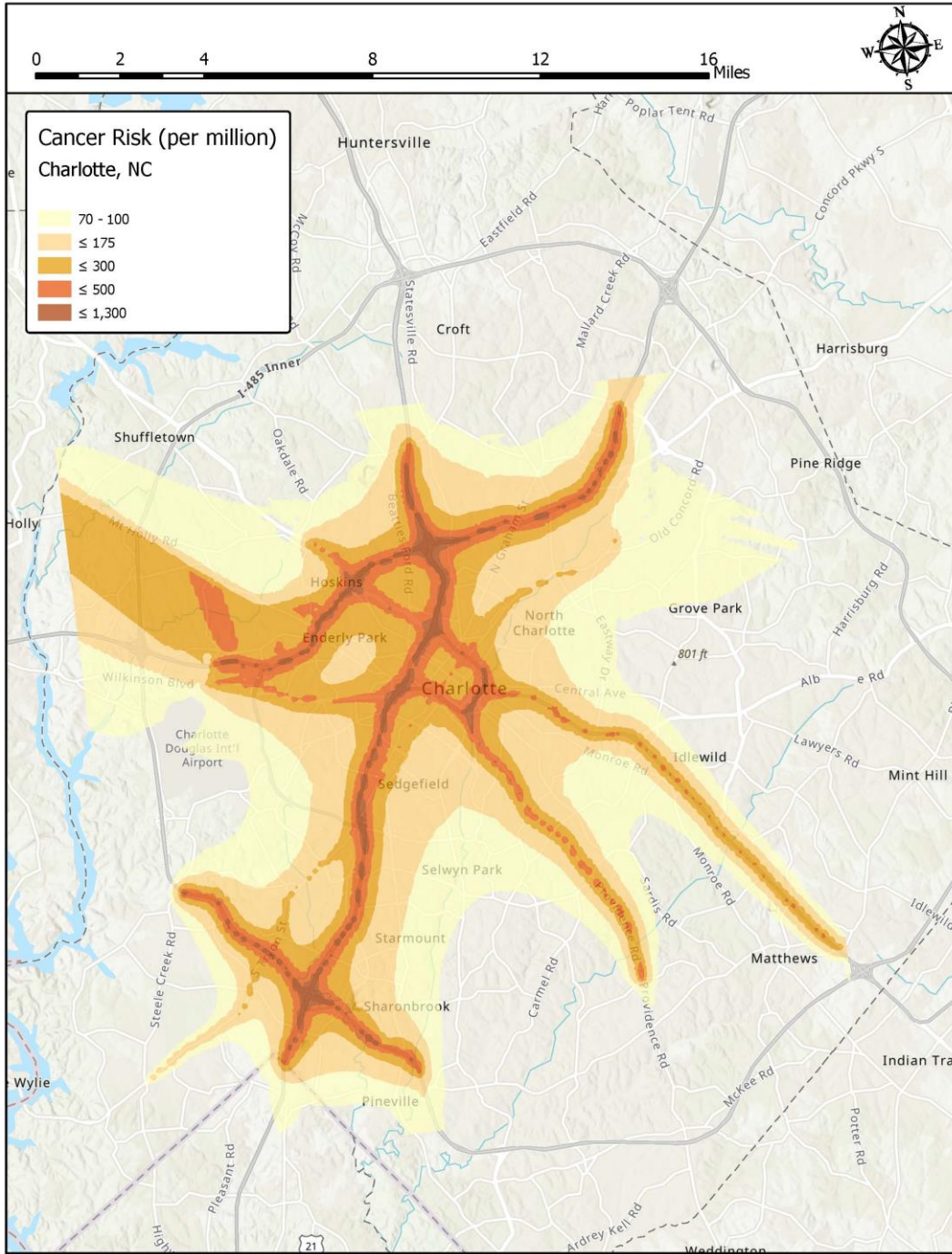
Table 6-24. Charlotte Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
I-77	I-77 – 130,700 AADT	20,759	59.7%
I-85	I-85 – 147,250 AADT	18,476	59.7%
R74	North Carolina Route 74 – 69,614 AADT	12,495	59.7%
NC16	North Carolina Route 16 – 71,210 AADT	10,116	59.7%
NC49	North Carolina Route 49 – 23,128 AADT	4,078	59.7%
I-485	I-485 – 129,400 AADT	9,584	59.7%

These sources were modeled with unit emission rates in AERMOD, and the Table 6-24 listed emission rates were input into CARB's HARP software to determine cancer risks from the DPM concentrations determined by AERMOD. While dispersion characteristics remained the same between baseline and reduced modeling scenarios, emission rates were reduced according to the number of "old technology" engines combusting diesel, based on source type. The table above shows the Proportion of "Old Technology" Engine Emissions where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

²⁸ <https://www.arcgis.com/apps/webappviewer/index.html?id=5f6fe58c1d90482ab9107ccc03026280>

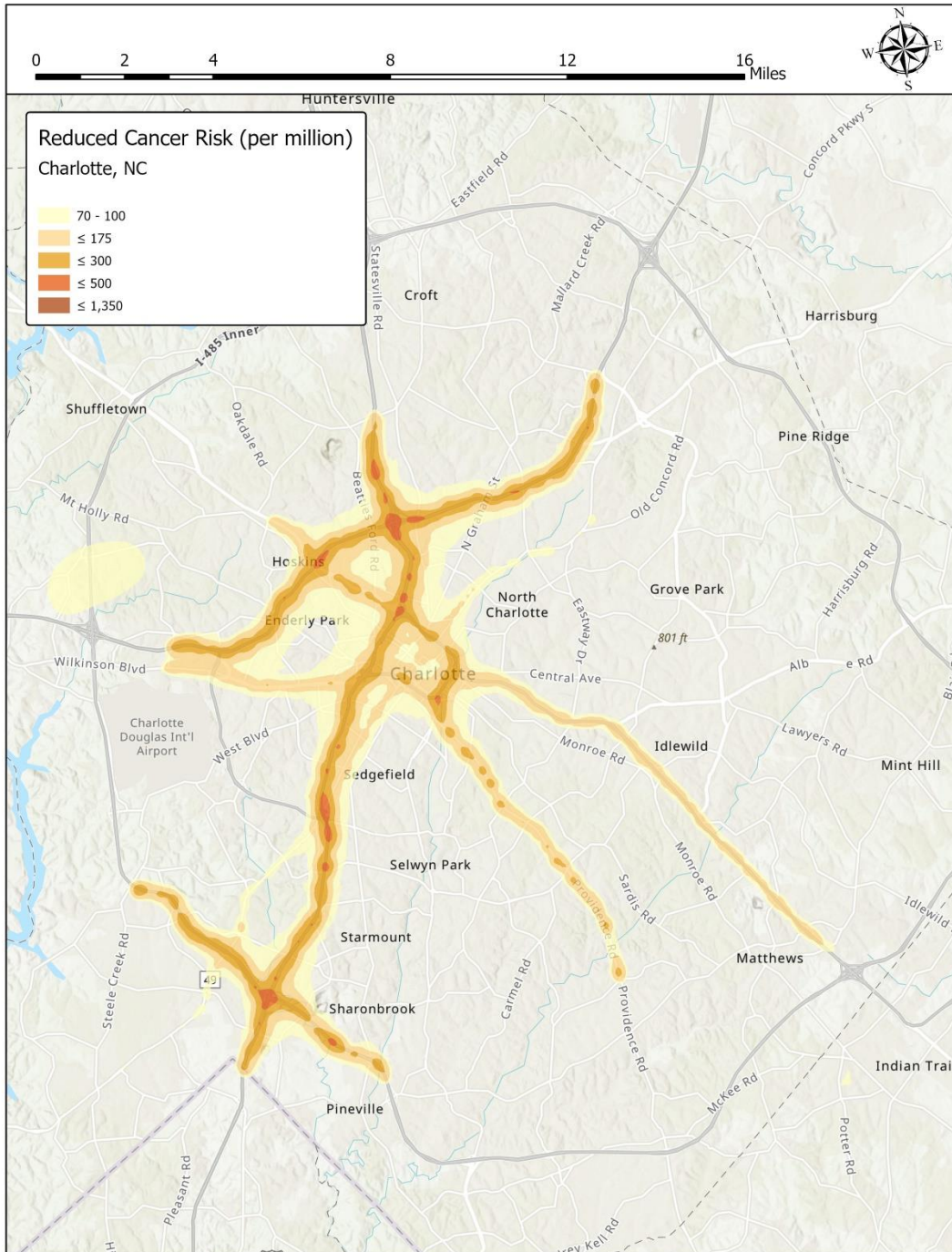
Figure 6-105. Charlotte Baseline Health Risk Assessment Isopleths



The site-specific HRA shows that the point of maximum impact (PMI) is substantially higher than the NATA/HARP evaluation, with an impact of 1,283 cancer cases per million residents. This PMI does not occur at a residential receptor, though, and does not represent an actual risk to residences in the area. The MEIR occurs at 511,330.9 m E, and 3,902,344.5 m N (NAD 83, UTM Zone 17), with a baseline risk of 946 cancer cases per million residents. This MEIR is higher than the NATA/HARP hybrid risks evaluated for that census tract (37119004500) with a total risk of 416 in a million. This HRA does not capture all of the cancer-causing

sources in the area but does demonstrate that NATA values are in-line with the site-specific demonstration with some extremely high local maxima due to local residences proximity to highways.

Figure 6-106. Charlotte Reduced Health Risk Assessment Isoleths



The reduced cancer risk PMI and MEIR are 517 and 381 in 1 million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 565 in 1 million at the MEIR.

6.11.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-25 below.

Table 6-25. Charlotte Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	29.4	\$982,748
Asthma Symptoms - Albuterol use	4,516	\$1,561
ER visits - All Cardiac Outcomes	3.6	\$4,160
ER visits – Respiratory	9.1	\$7,934
HA – All – Respiratory	1.1	\$17,318
HA – Alzheimer’s Disease	2.8	\$33,719
HA – Cardio Cerebro- and Peripheral Vascular Disease	1.2	\$18,322
HA – Parkinson’s Disease	0.5	\$6,314
HA – Respiratory-2	0.2	\$0
HA – Respiratory-2 HA – All Respiratory	1.3	\$0
Incidence – Asthma	35.5	\$1,586,374
Incidence – Hay Fever/Rhinitis	218.8	\$131,241
Incidence – Lung Cancer	1.4	\$17,312
Incidence – Out of Hospital Cardiac Arrest	0.2	\$6,128
Incidence – Stroke	0.5	\$17,847
Minor Restricted Activity Days	10,239	\$712,440
Mortality – All Cause	8.5	\$66,137,090
Work Loss Days	1,751	\$309,998
Total		\$69,990,503

6.12 Cleveland, Ohio

6.12.1 NATA Health Risks

The subsections below review the NATA data available for the Cleveland, OH (Cleveland) community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-107 shows the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

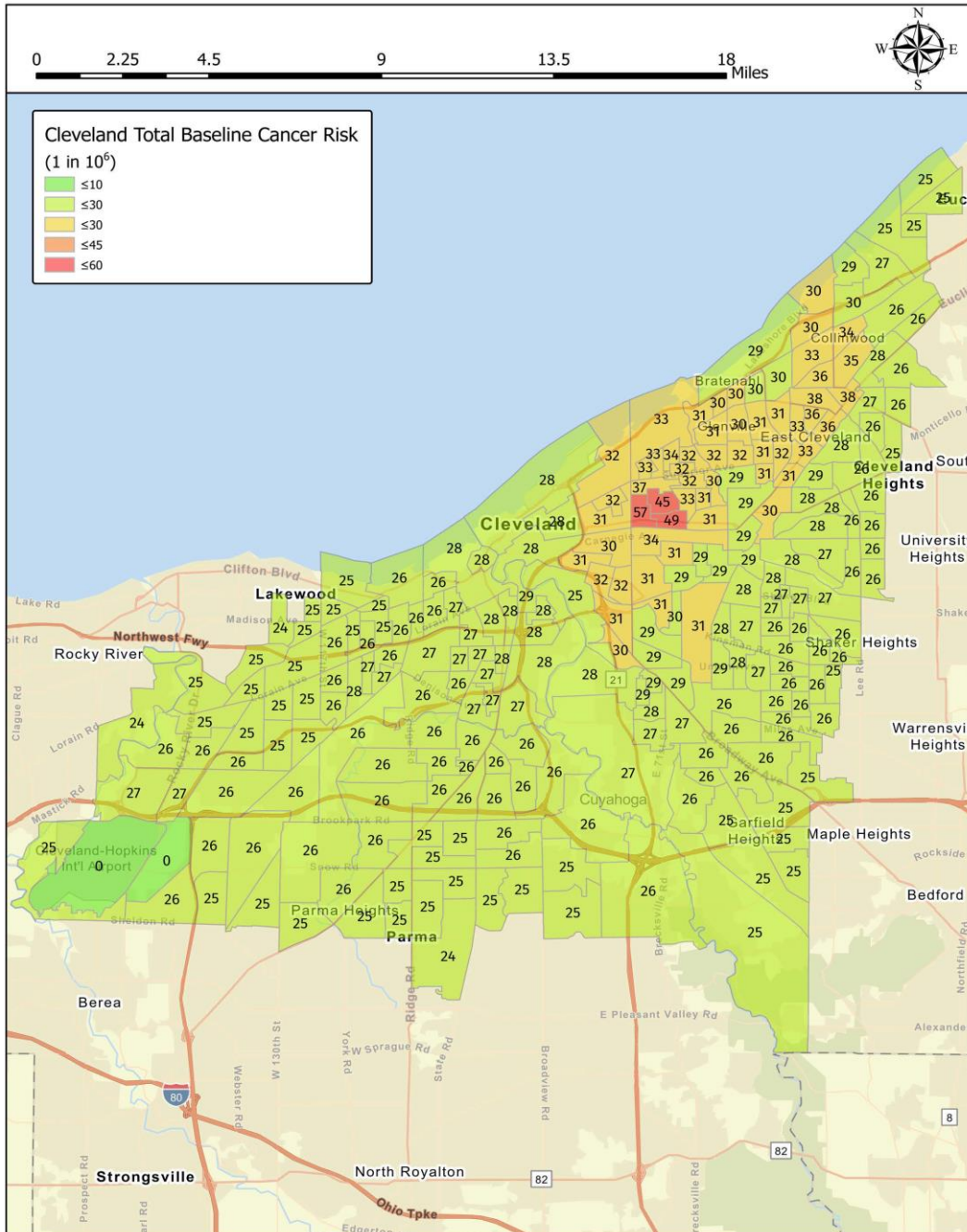
Figure 6-108 shows those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-109 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Cleveland community.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the Cleveland community.

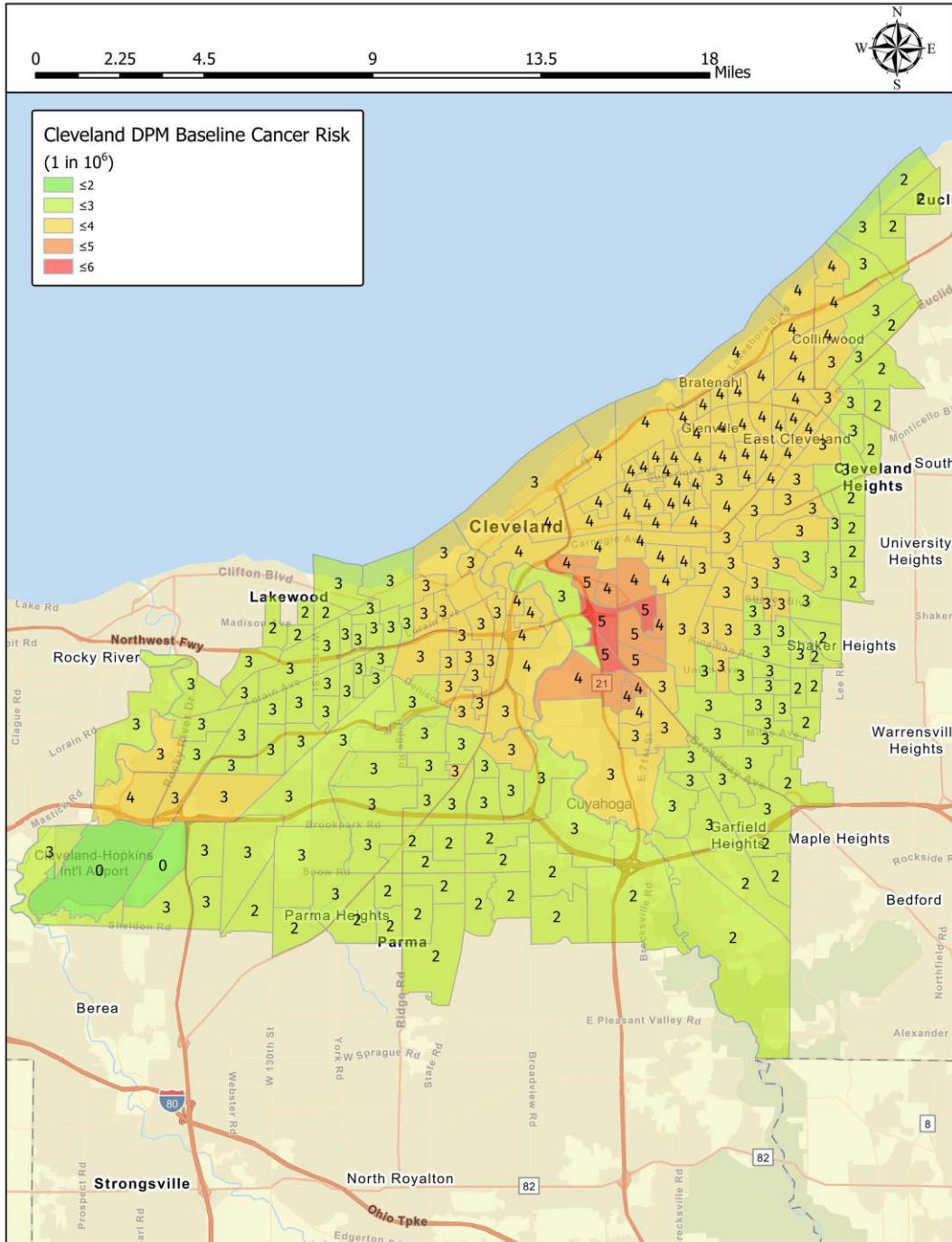
6.12.1.1 NATA Risk Data

Figure 6-107. Cleveland Baseline NATA Total Cancer Risks



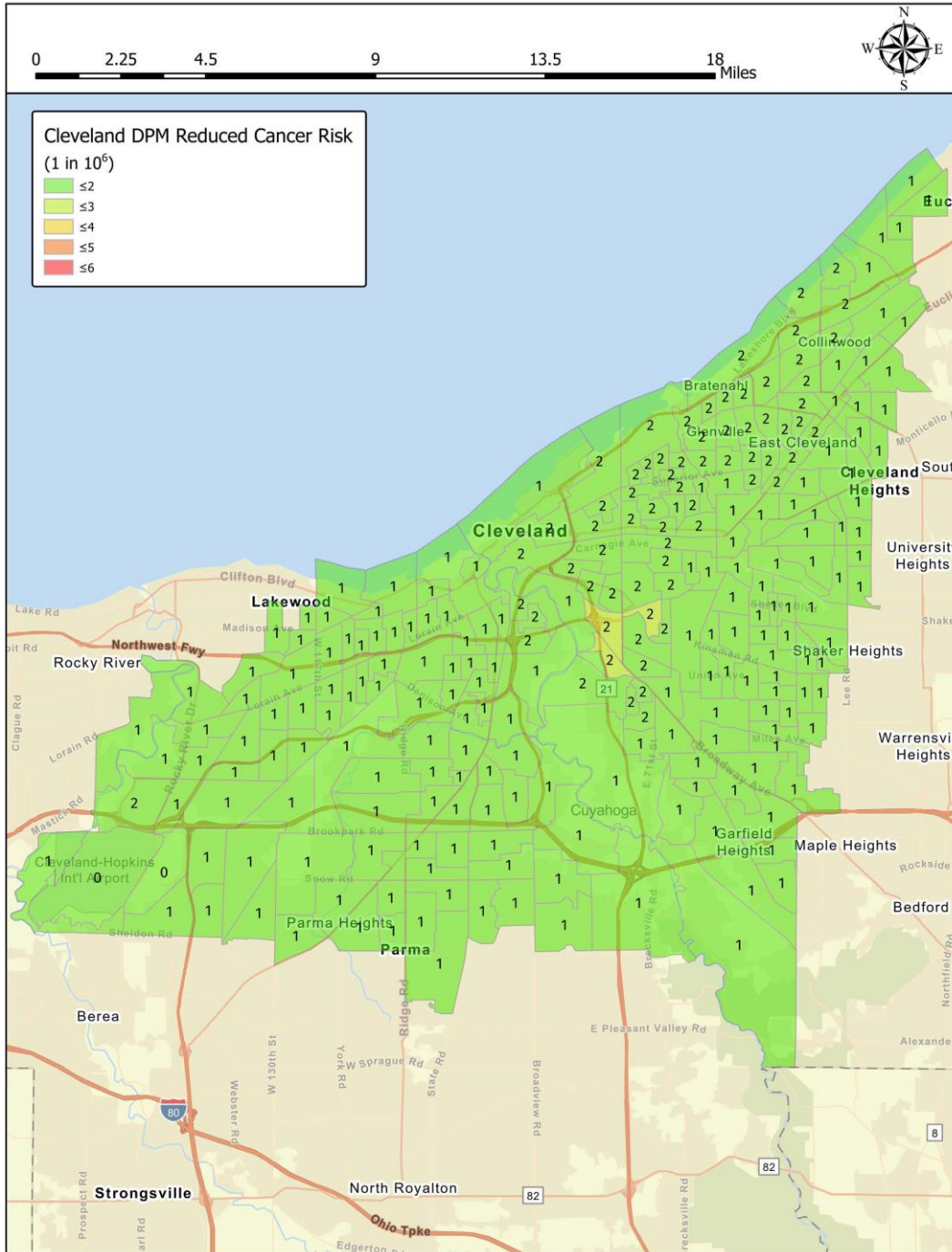
According to the NATA, the maximum baseline cancer risk in the Cleveland community is 57.16 cancer cases per million residents for census tract 39035112301, with a population of 1,427 residents. When accounting for all of the communities assessed, the total cancer burden for the Cleveland community is 16 cancer cases expected over a 70-year timeline among a total community population of 599,385.

Figure 6-108. Cleveland Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the Cleveland community is 5 cancer cases per million residents for census tract 39035110501, with a population of 851 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Cleveland community is 2 cancer cases expected over a 70-year timeline among a total community population of 599,385.

Figure 6-109. Cleveland Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Cleveland community becomes 2 cancer cases per million residents for census tract 39035110501, with a population of 851 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Cleveland community becomes 1 cancer case expected over a 70-year timeline among a total community population of 599,385.

6.12.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB's HARP program with OEHHA cancer unit risk values. The data is outlined in the following order:**

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program.**

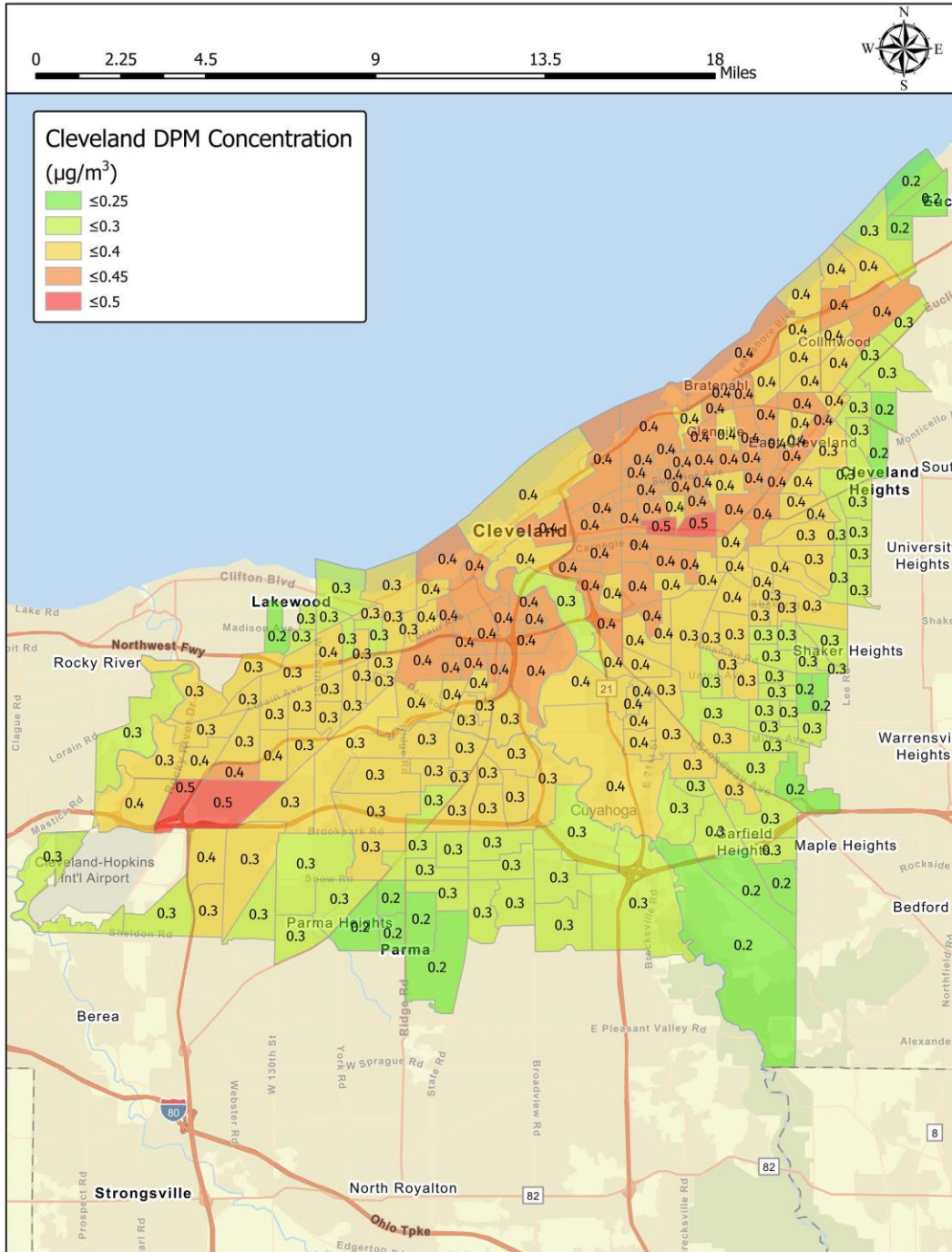
Figure 6-110 shows the baseline DPM concentrations provided by the NATA.

Figure 6-111 shows the baseline DPM-specific cancer risks as determined using the NATA concentration **values and CARB's HARP program.**

Figure 6-112 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Cleveland community.

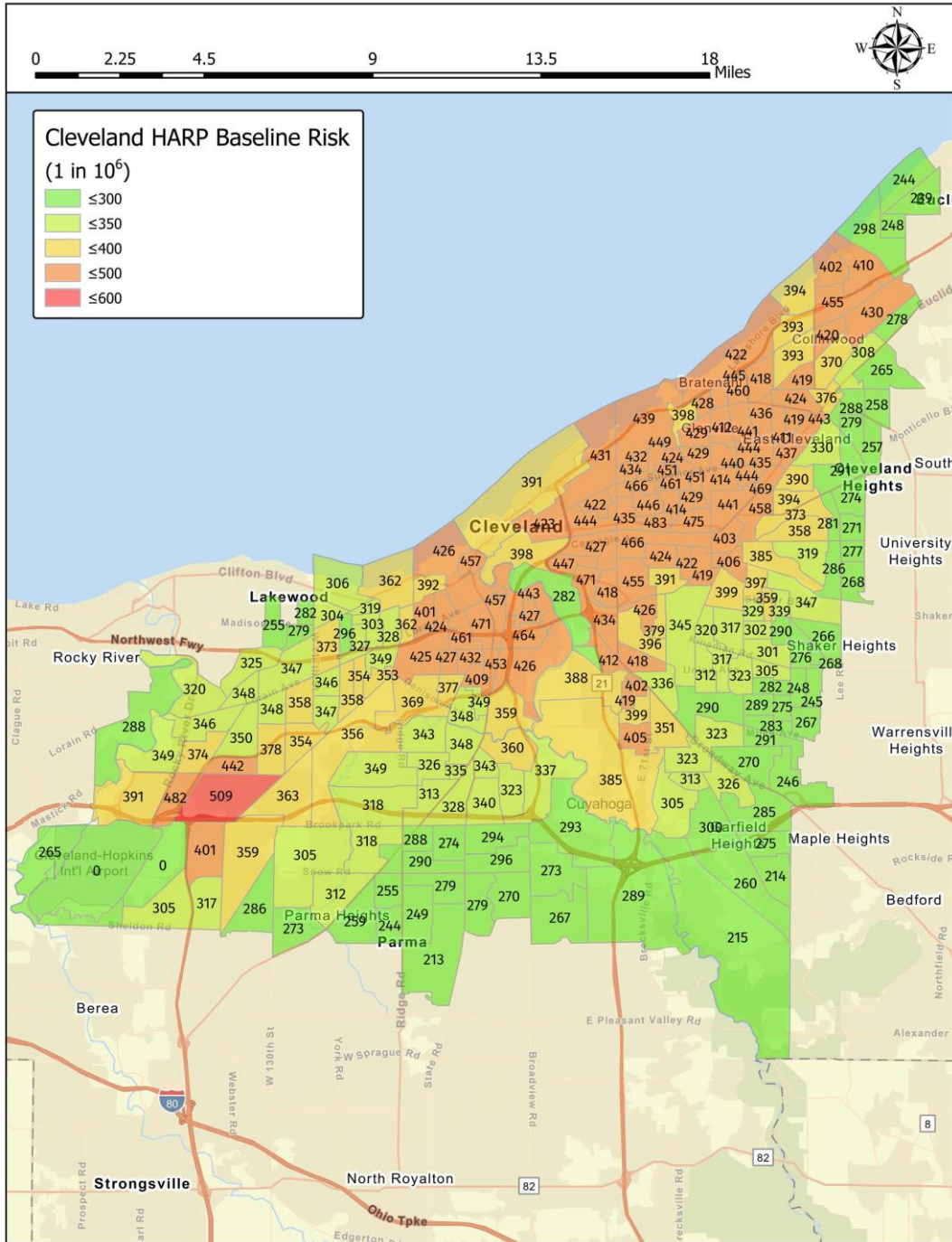
Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the Cleveland community.

Figure 6-110. Cleveland Baseline NATA DPM Concentrations



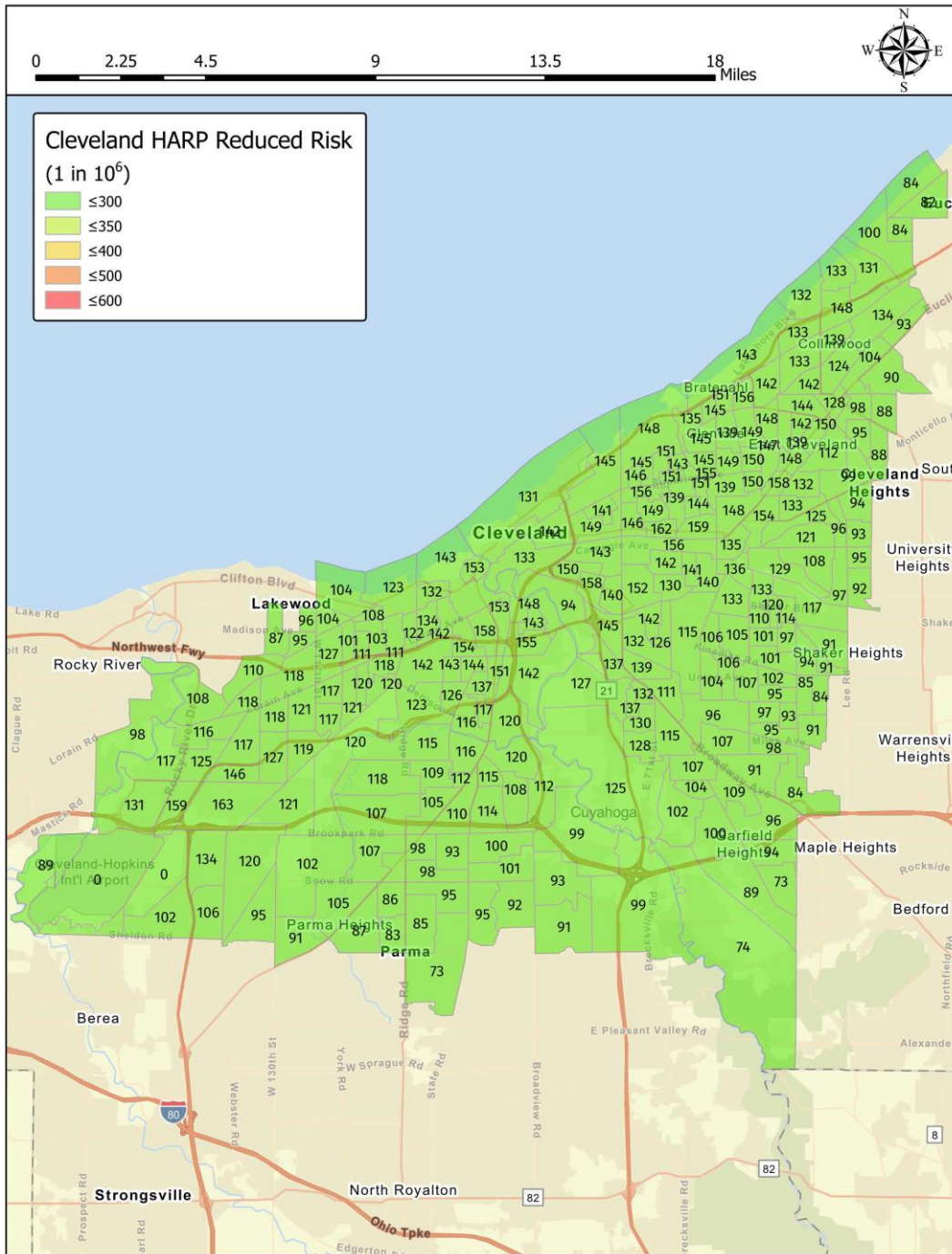
According to the NATA, the maximum baseline DPM concentration in the Cleveland community is $0.48 \mu\text{g}/\text{m}^3$ for census tract 39035124500, with a population of 3,456 residents. The average DPM concentration of the Cleveland community is $0.34 \mu\text{g}/\text{m}^3$.

Figure 6-111. Cleveland Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the Cleveland community is 509 cancer cases per million residents for census tract 39035124500, with a population of 3,456 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Cleveland community is 208 cancer cases expected over a 70-year timeline among a total community population of 599,385.

Figure 6-112. Cleveland Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Cleveland community becomes 163 cancer cases per million residents for census tract 39035124500, with a population of 3,456 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Cleveland community becomes 70 cancer cases expected over a 70-year timeline among a total community population of 599,385.

6.12.2 Cleveland Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Cleveland. The following sources were utilized to generate the HRA.

- Ohio Department of Transportation (ODOT) – Traffic Counts (2019 Average Annual Daily Traffic)²⁹

The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-11.

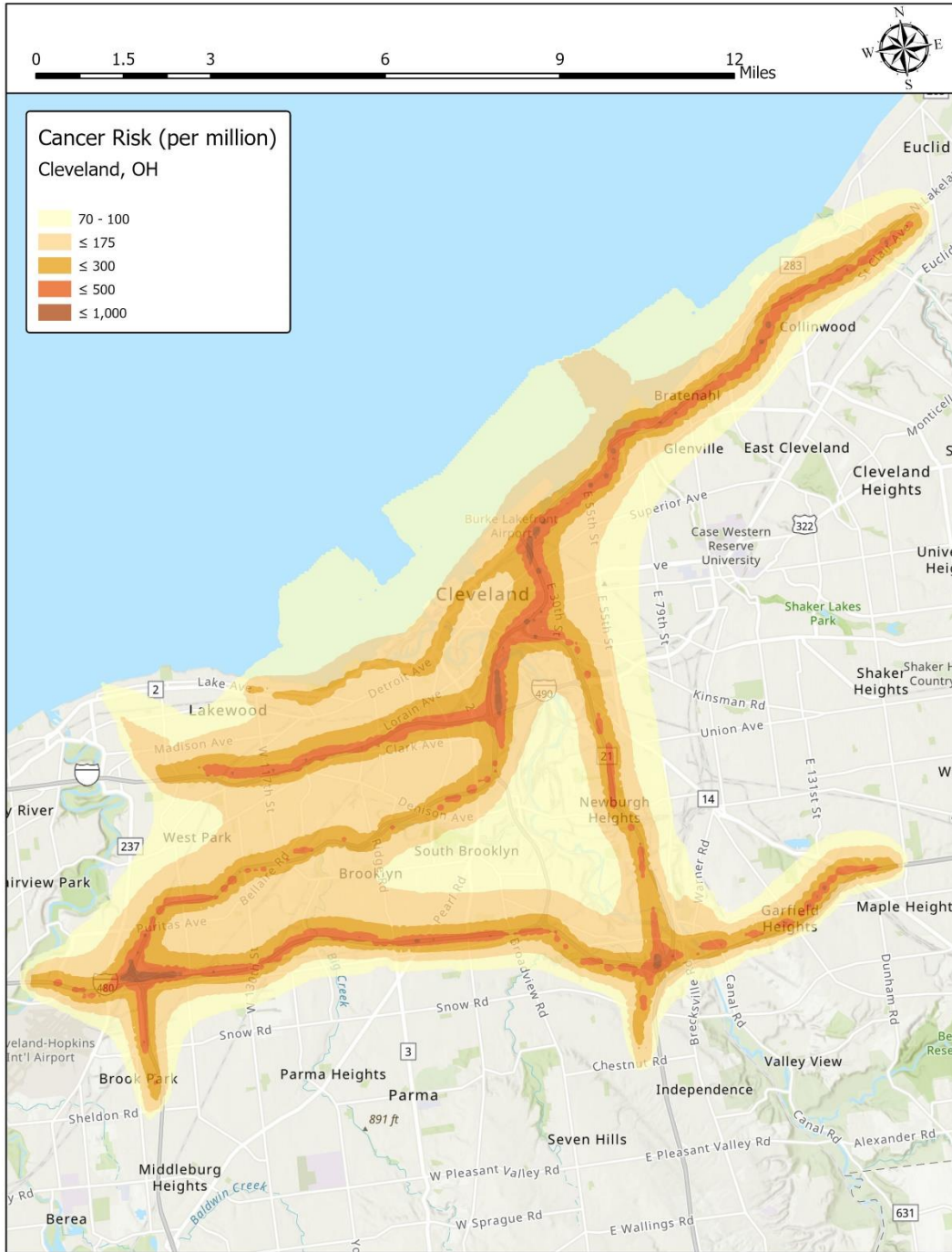
Table 6-26. Cleveland Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
I-90	I-90 – 131,467 AADT	24,146	59.7%
I-480	I-480 – 134,259 AADT	21,467	59.7%
I-77	I-77 – 84,152 AADT	7,239	59.7%
I-71	I-71 – 85,187 AADT	9,497	59.7%
Cle Mem Shrwy	Cleveland Memorial Shoreway – 50,712 AADT	3,246	59.7%

These sources were modeled with unit emission rates in AERMOD, and the Table 6-11. listed emission rates **were input into CARB's HARP software to determine cancer risks from the DPM concentrations determined by AERMOD.** While dispersion characteristics remained the same between baseline and reduced modeling scenarios, **emission rates were reduced according to the number of "old technology" engines combusting diesel, based on source type.** The table above shows **the Proportion of "Old Technology" Engine Emissions** where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

²⁹ <https://www.njtms.org/map/>

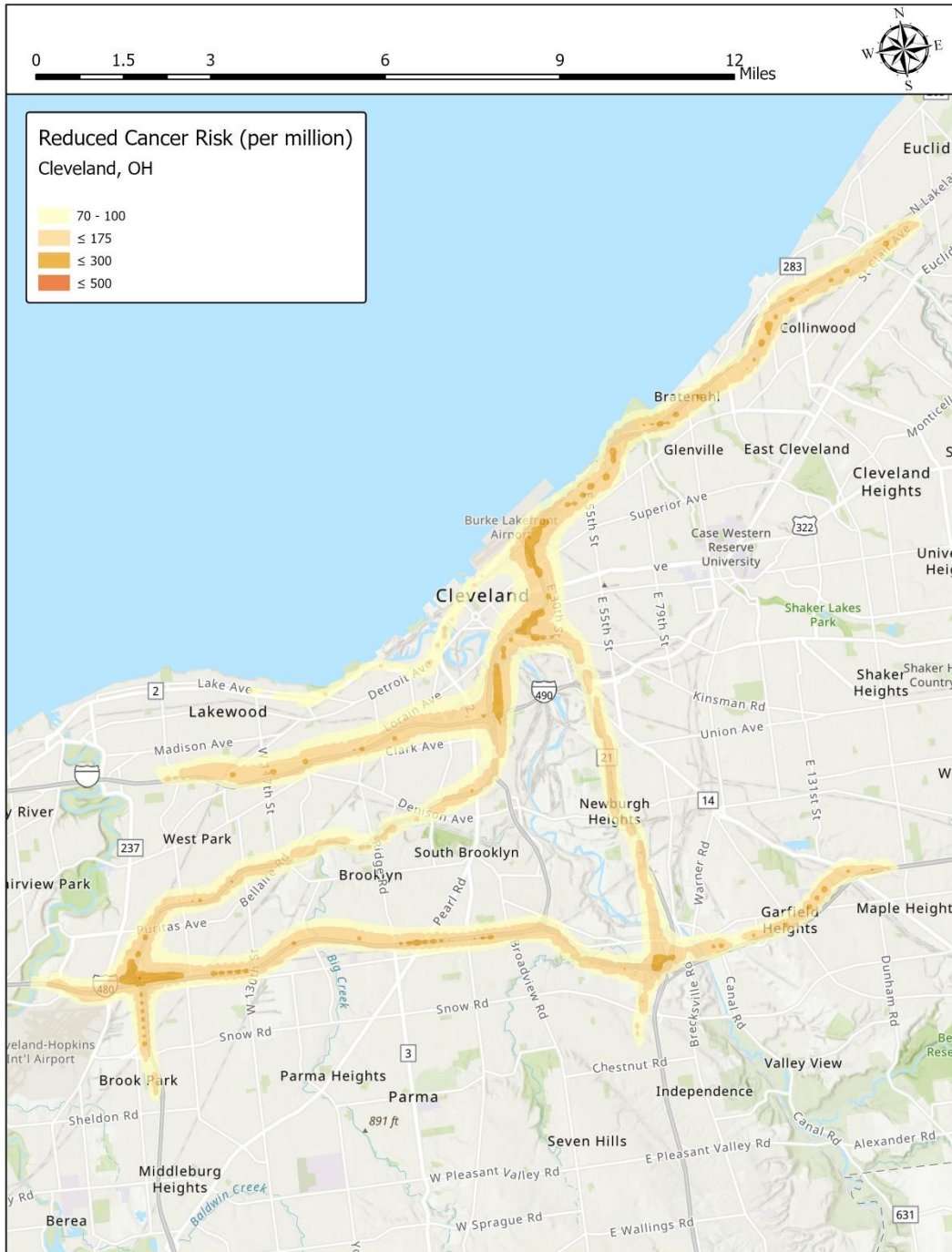
Figure 6-113. Cleveland Baseline Health Risk Assessment Isopleths



The site-specific HRA shows that the point of maximum impact (PMI) is substantially higher than the NATA/HARP evaluation, with an impact of 835 cancer cases per million residents. This PMI does not occur at a residential receptor, though, and does not represent an actual risk to residences in the area. The MEIR occurs at 951,523.1 m E, and 4,615,231.4 m N (NAD 83, UTM Zone 17), with a baseline risk of 771 cancer cases per million residents. This MEIR is higher than the NATA/HARP hybrid risks evaluated for that census tract (39035117102) with a total risk of 393 in a million. This HRA does not capture all of the cancer-causing

sources in the area but does demonstrate that NATA values are in-line with the site-specific demonstration with some extremely high local maxima due to local residences proximity to highways.

Figure 6-114. Cleveland Reduced Health Risk Assessment Isoleths



The reduced cancer risk PMI and MEIR are 336 and 311 in 1 million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 460 in 1 million at the MEIR.

6.12.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-27 below.

Table 6-27. Cleveland Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	34.8	\$1,089,128
Asthma Symptoms - Albuterol use	3,414	\$1,180
ER visits - All Cardiac Outcomes	3.7	\$4,294
ER visits – Respiratory	7.0	\$6,165
HA – All – Respiratory	0.8	\$17,751
HA – Alzheimer’s Disease	6.1	\$73,390
HA – Cardio Cerebro- and Peripheral Vascular Disease	1.7	\$25,994
HA – Parkinson’s Disease	0.8	\$10,135
HA – Respiratory-2	0.3	\$0
HA – Respiratory-2 HA – All Respiratory	1.1	\$0
Incidence – Asthma	25.3	\$1,130,525
Incidence – Hay Fever/Rhinitis	162.6	\$97,545
Incidence – Lung Cancer	1.6	\$19,794
Incidence – Out of Hospital Cardiac Arrest	0.2	\$6,318
Incidence – Stroke	0.7	\$22,970
Minor Restricted Activity Days	7,963	\$554,102
Mortality – All Cause	13.9	\$108,813,863
Work Loss Days	1,345	\$234,697
Total		\$112,107,852

6.13 Philadelphia-Reading-Camden (NJ), Pennsylvania

6.13.1 NATA Health Risks

The subsections below review the NATA data available for the Philadelphia-Reading-Camden, PA/NJ (Philadelphia) community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-115 and Figure 6-116 shows the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

Figure 6-117 and Figure 6-118 shows those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-119 and Figure 6-120 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Philadelphia community.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the Philadelphia community.

6.13.1.1 NATA Risk Data

Figure 6-115. S. Philadelphia Baseline NATA Total Cancer Risks

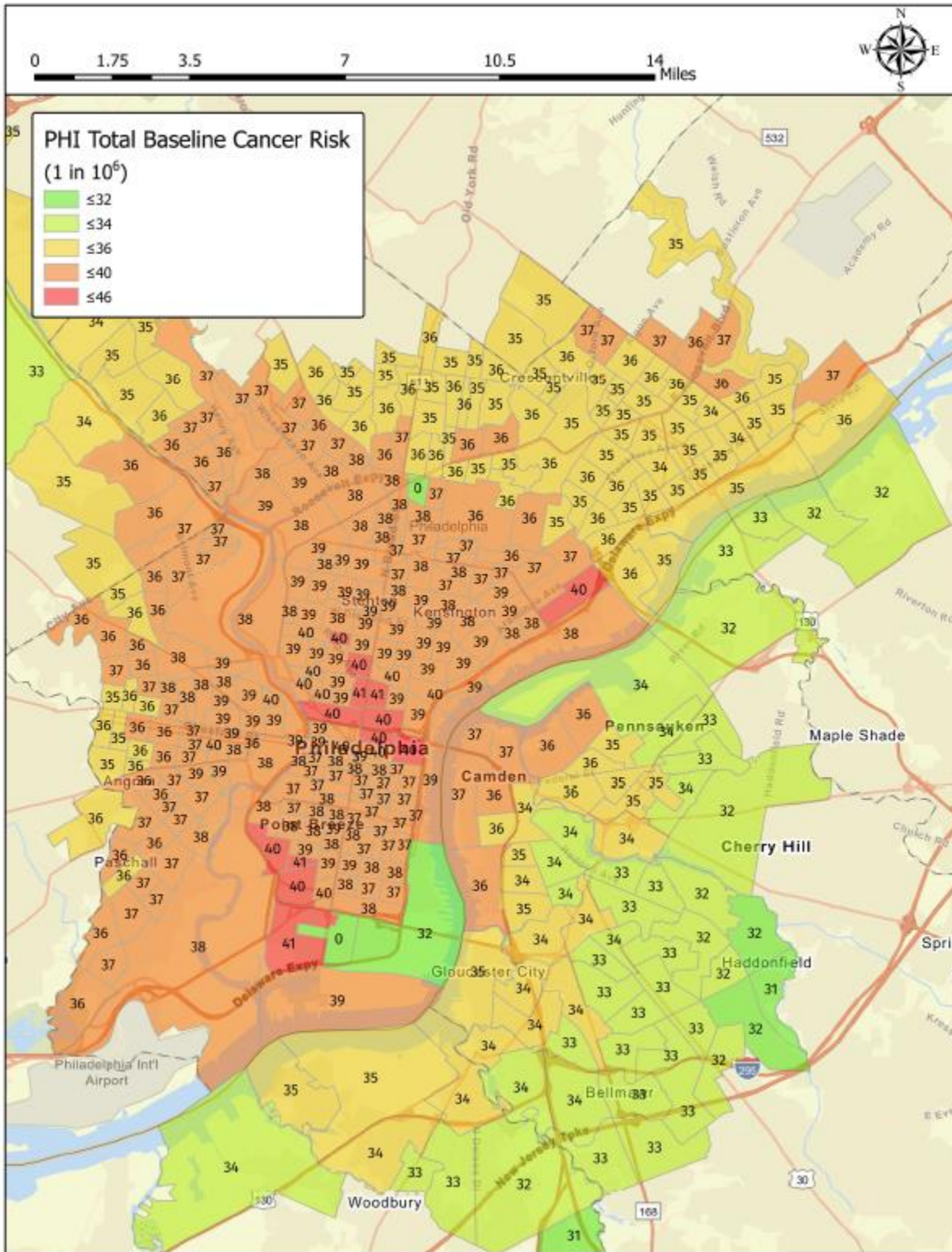
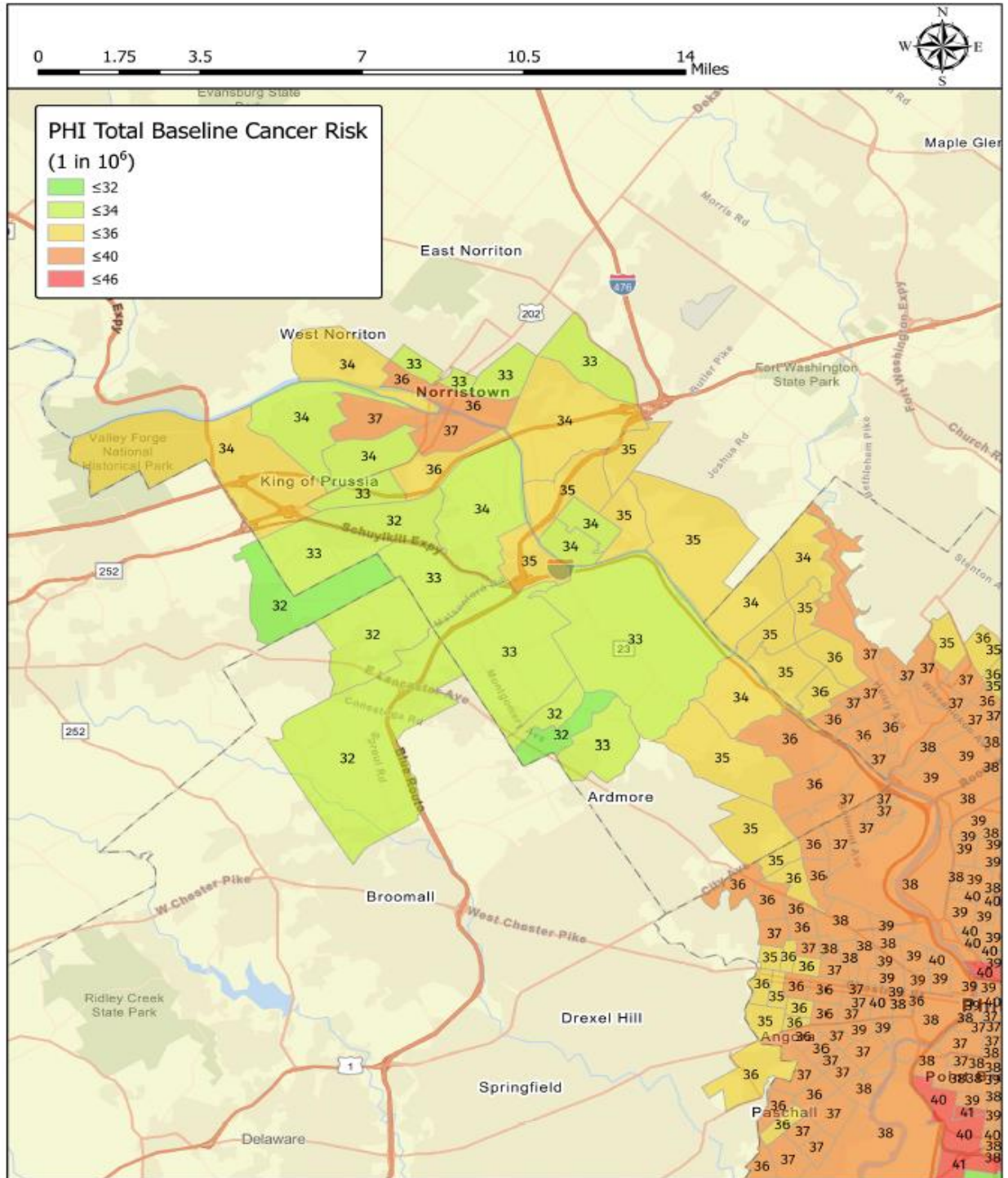


Figure 6-116. N. Philadelphia Baseline NATA Total Cancer Risks



According to the NATA, the maximum baseline cancer risk in the Philadelphia community is 41.21 cancer cases per million residents for census tract 42101037300, with a population of 5,137 residents. When accounting for all of the communities assessed, the total cancer burden for the Philadelphia community is 61 cancer cases expected over a 70-year timeline among a total community population of 1,670,574.

Figure 6-117. S. Philadelphia Baseline NATA DPM Cancer Risks

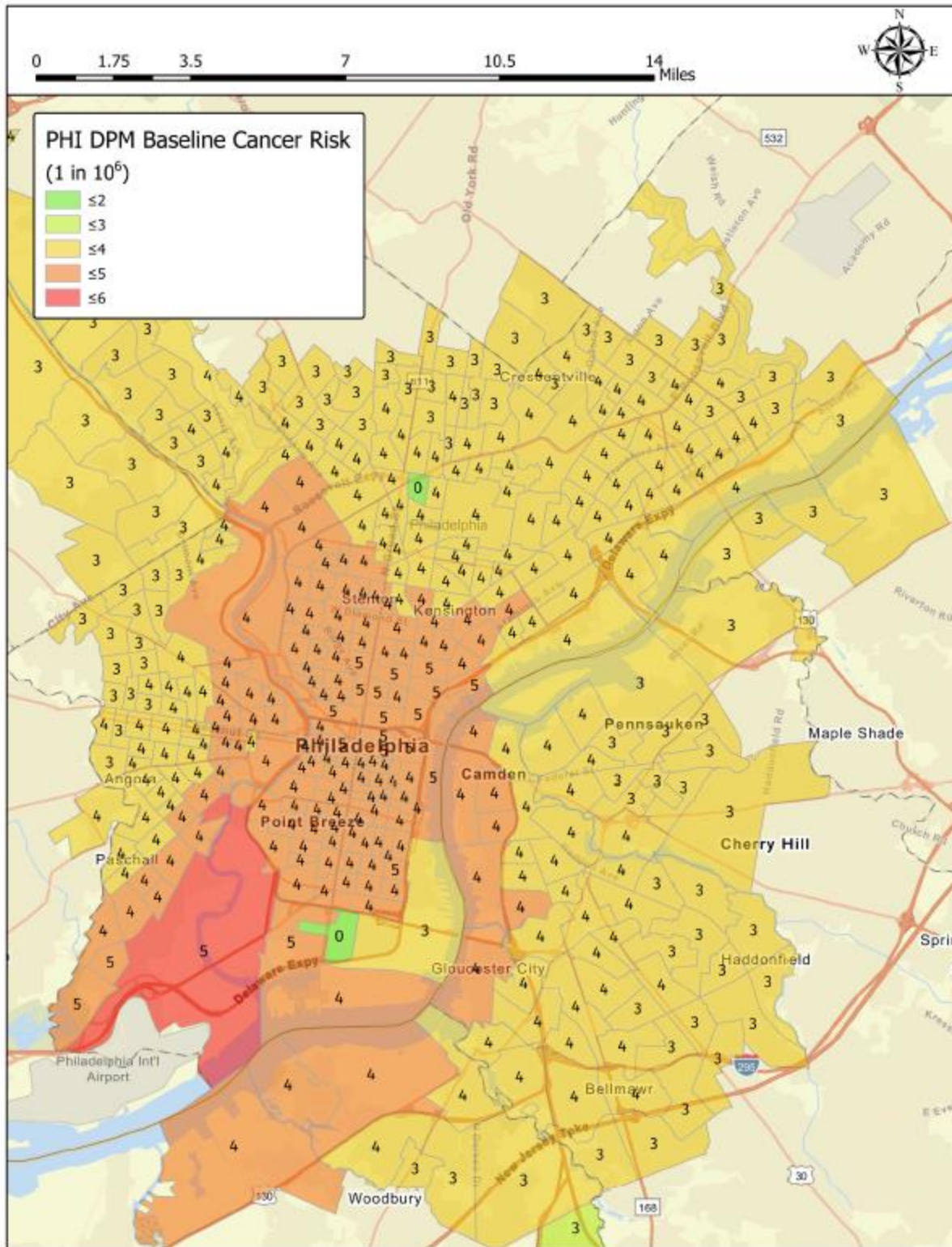
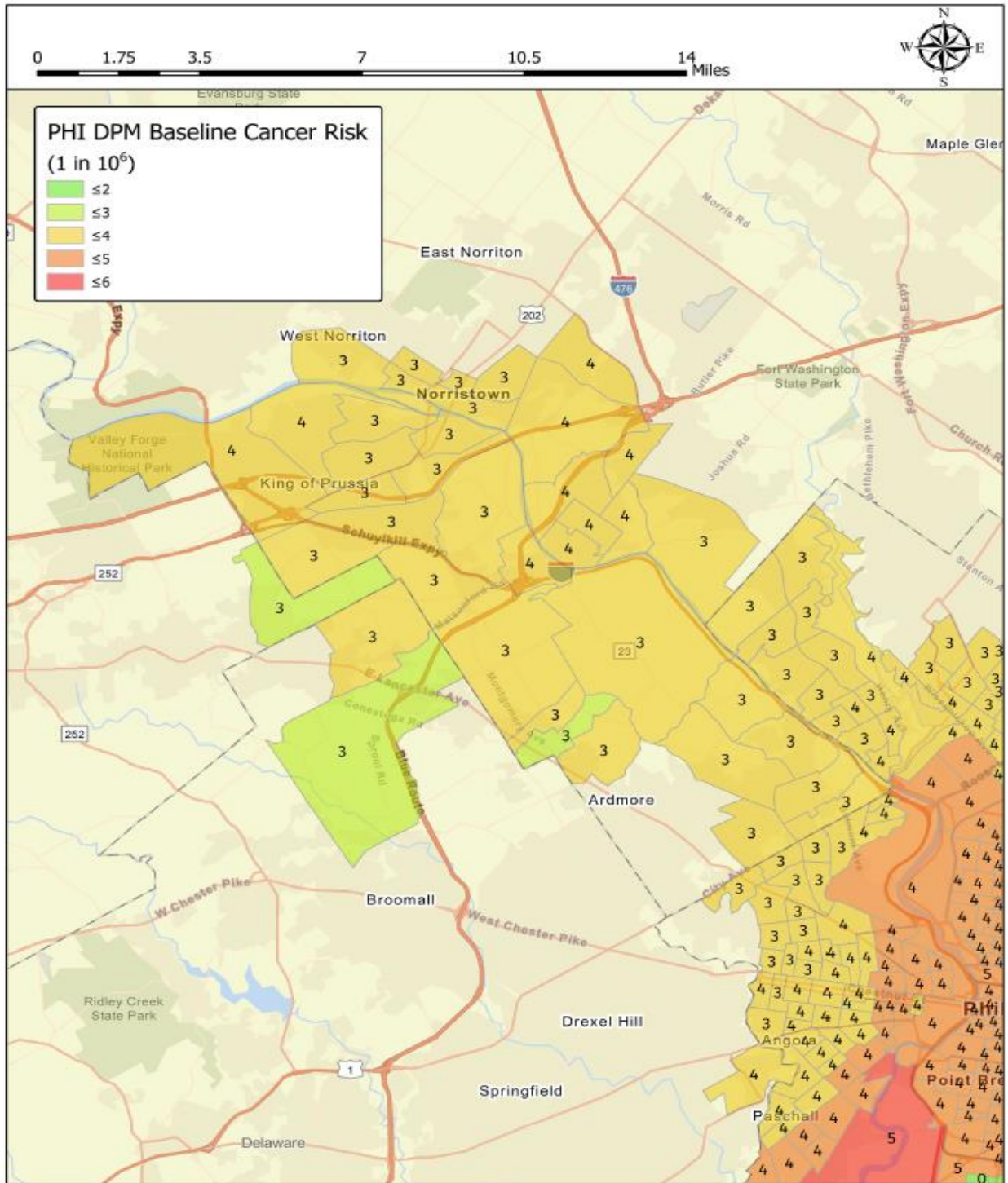


Figure 6-118. N. Philadelphia Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the Philadelphia community is 5.25 cancer cases per million residents for census tract 42101980900, with a population of 30 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Philadelphia community is 6 cancer cases expected over a 70-year timeline among a total community population of 1,670,574.

Figure 6-119. S. Philadelphia Reduced NATA DPM Cancer Risks

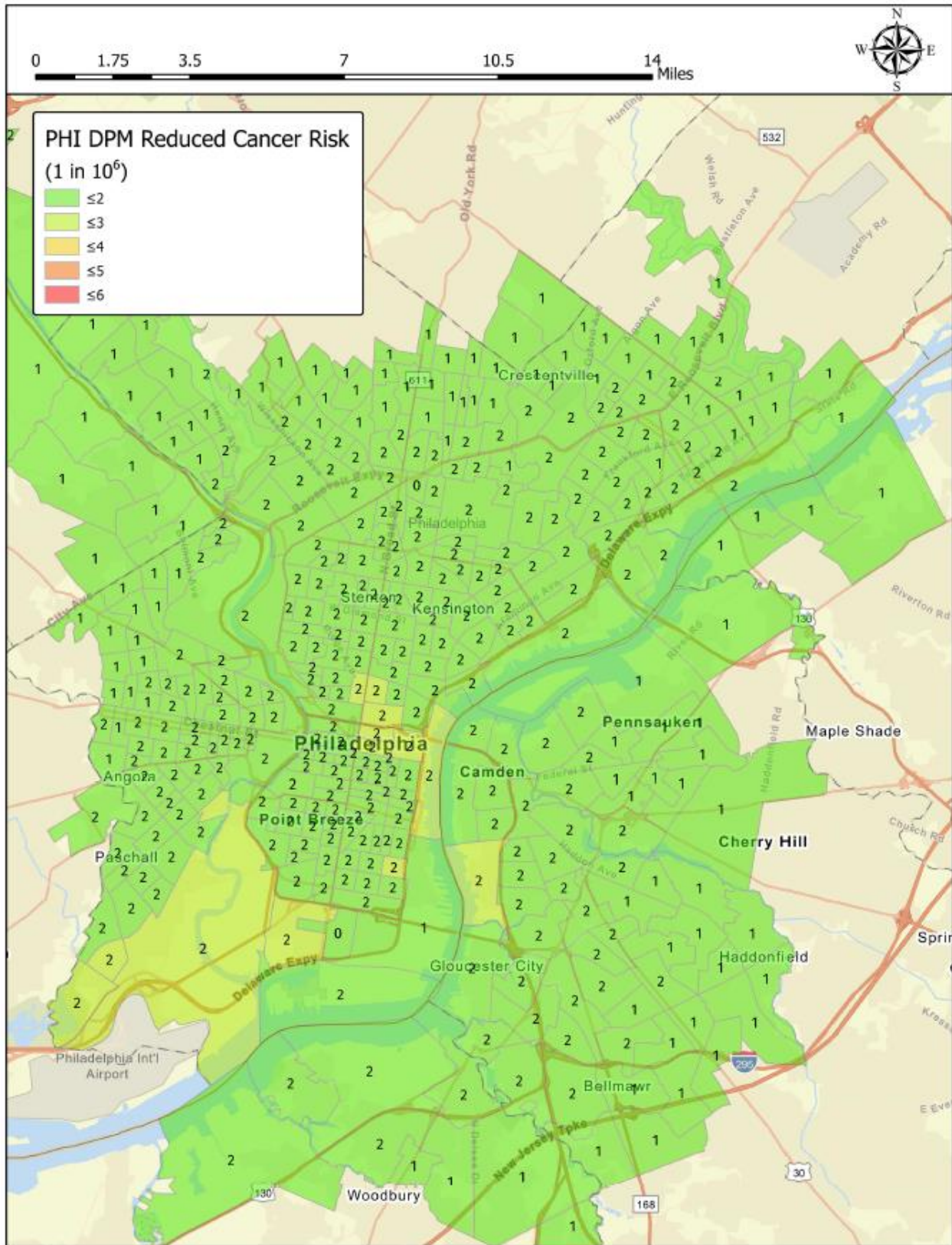
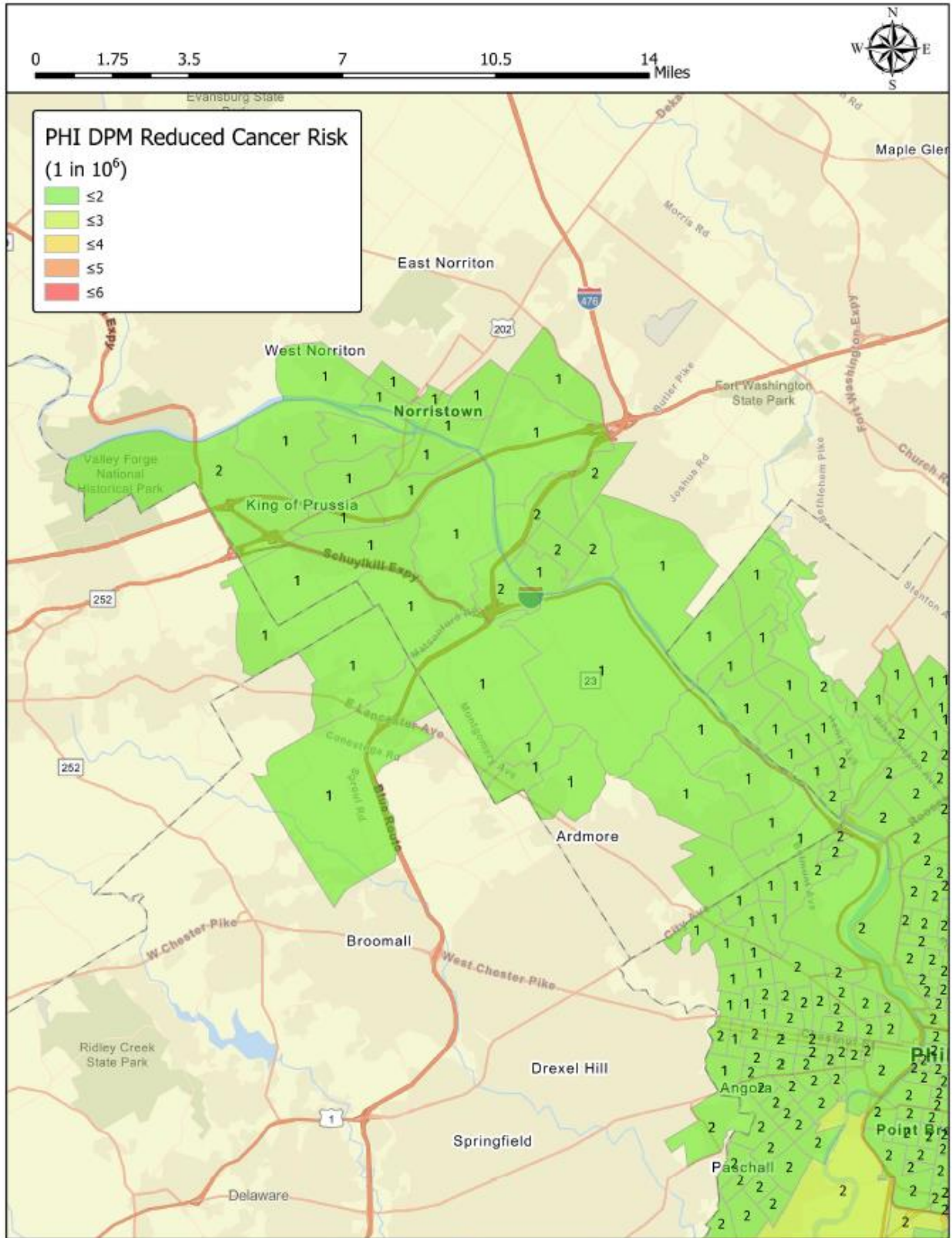


Figure 6-120. N. Philadelphia Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Philadelphia community becomes 2.3 cancer cases per million residents for census tract 42101980900, with a population of 30 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Philadelphia community becomes 3 cancer cases expected over a 70-year timeline among a total community population of 1,670,574.

6.13.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB's HARP program with OEHHA cancer unit risk values. The data is outlined in the following order:**

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program.**

Figure 6-121 and Figure 6-122 shows the baseline DPM concentrations provided by the NATA.

Figure 6-123 and Figure 6-124 shows the baseline DPM-specific cancer risks as determined using the NATA **concentration values and CARB's HARP program.**

Figure 6-125 and Figure 6-125 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Philadelphia community.

Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the Philadelphia community.

Figure 6-121. S. Philadelphia Baseline NATA DPM Concentrations

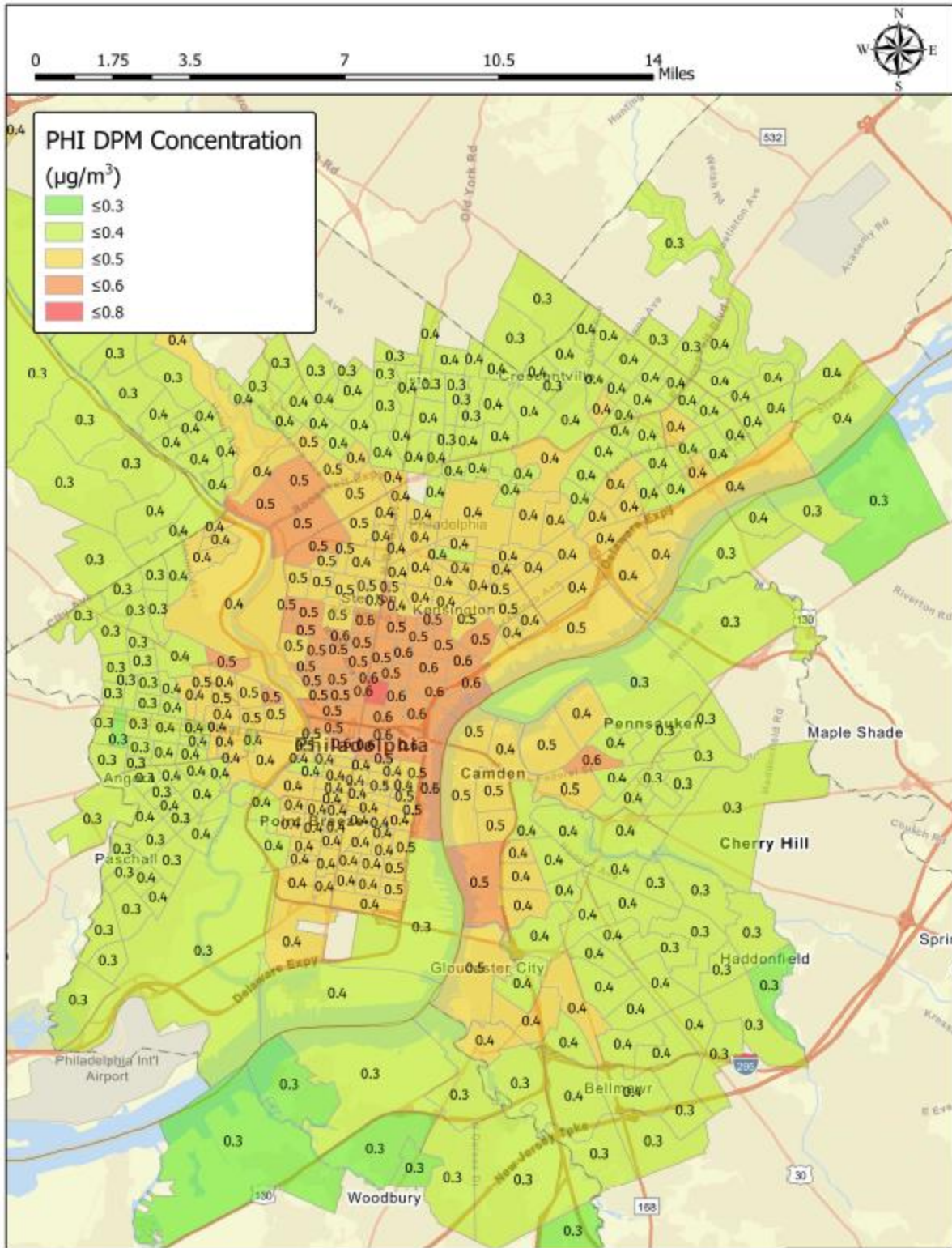
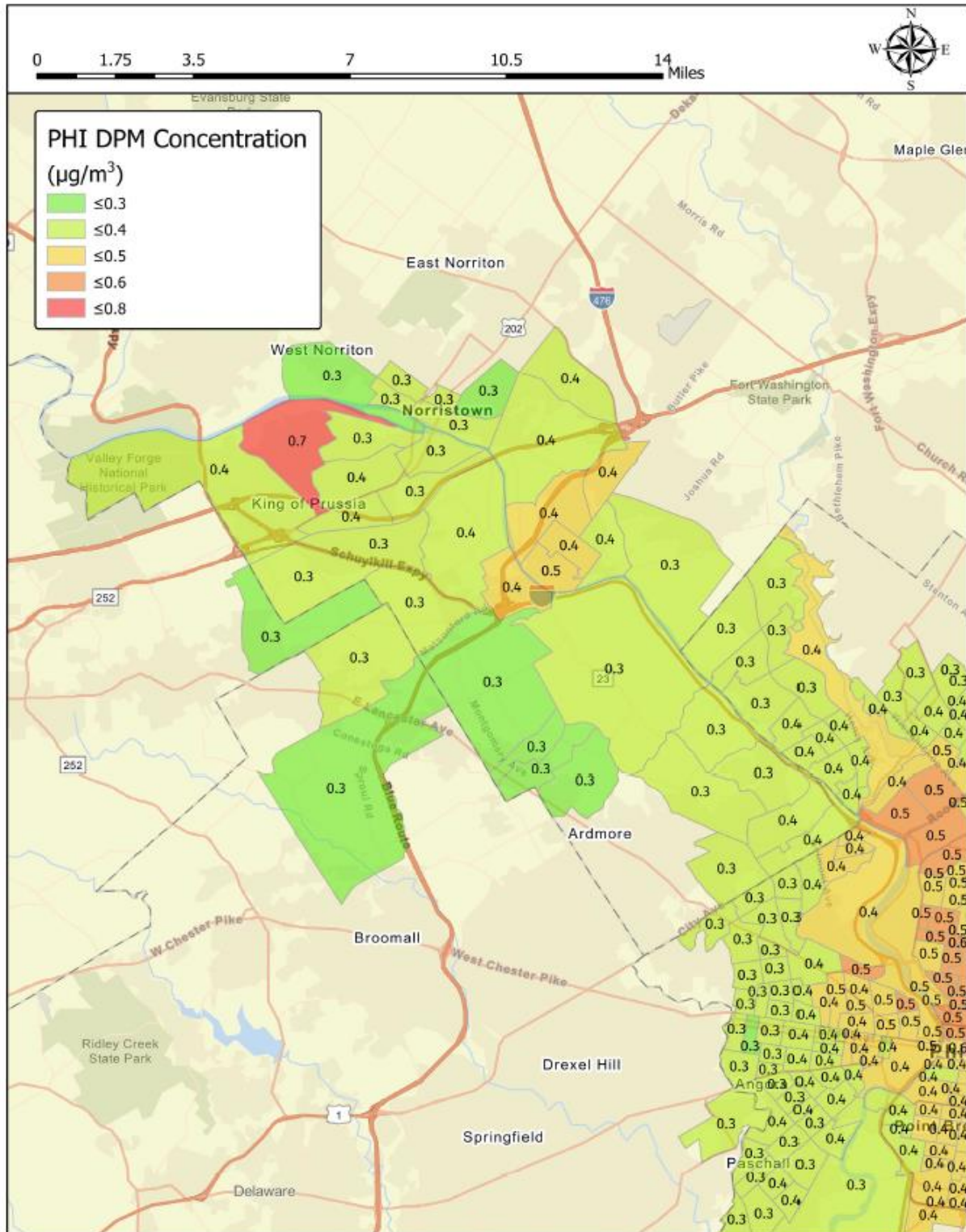


Figure 6-122. N. Philadelphia Baseline NATA DPM Concentrations



According to the NATA, the maximum baseline DPM concentration in the Philadelphia community is 0.67 $\mu\text{g}/\text{m}^3$ for census tract 42091205808, with a population of 5,340 residents. The average DPM concentration of the Philadelphia community is 0.40 $\mu\text{g}/\text{m}^3$.

Figure 6-123. S. Philadelphia Baseline NATA/HARP DPM Hybrid Risks

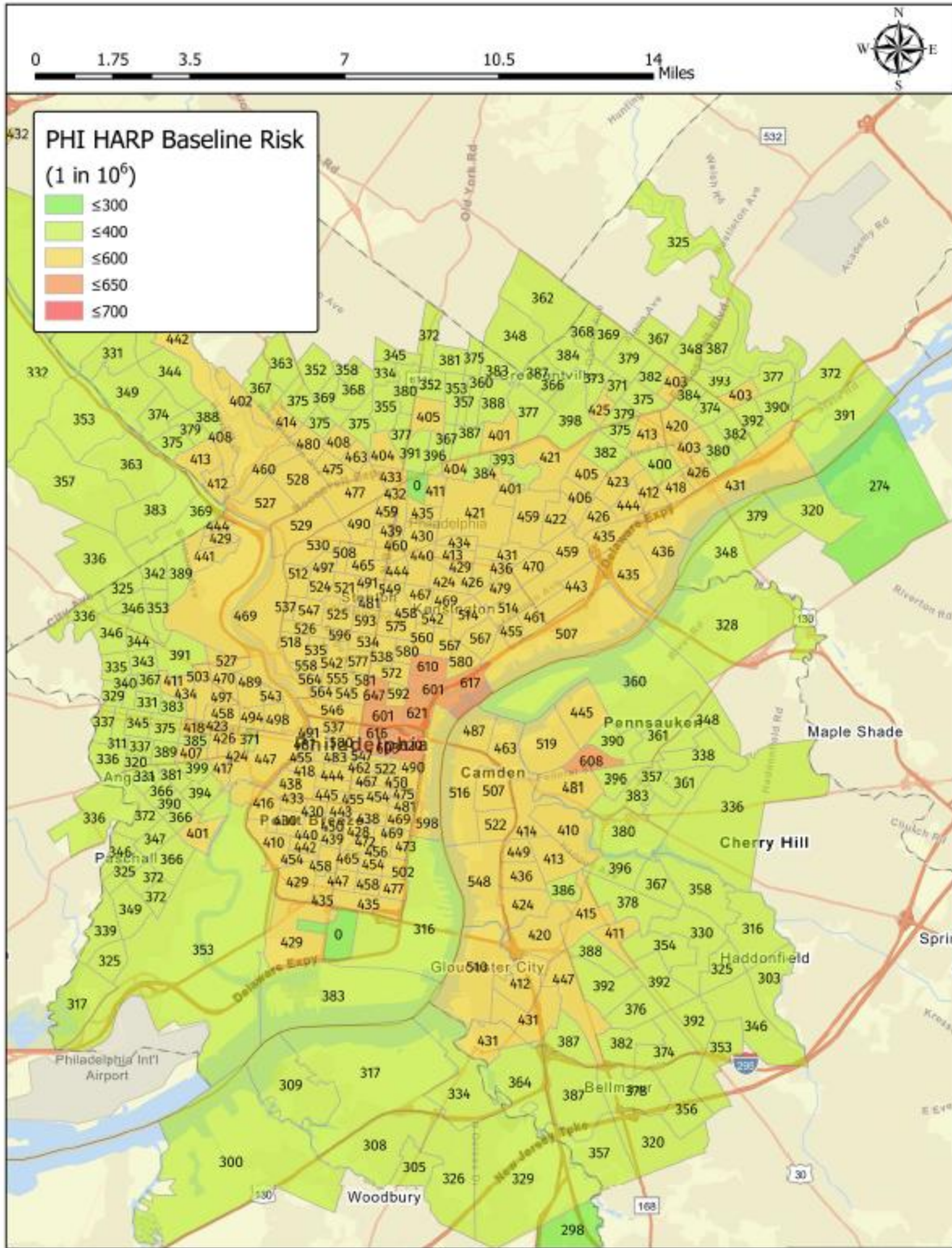
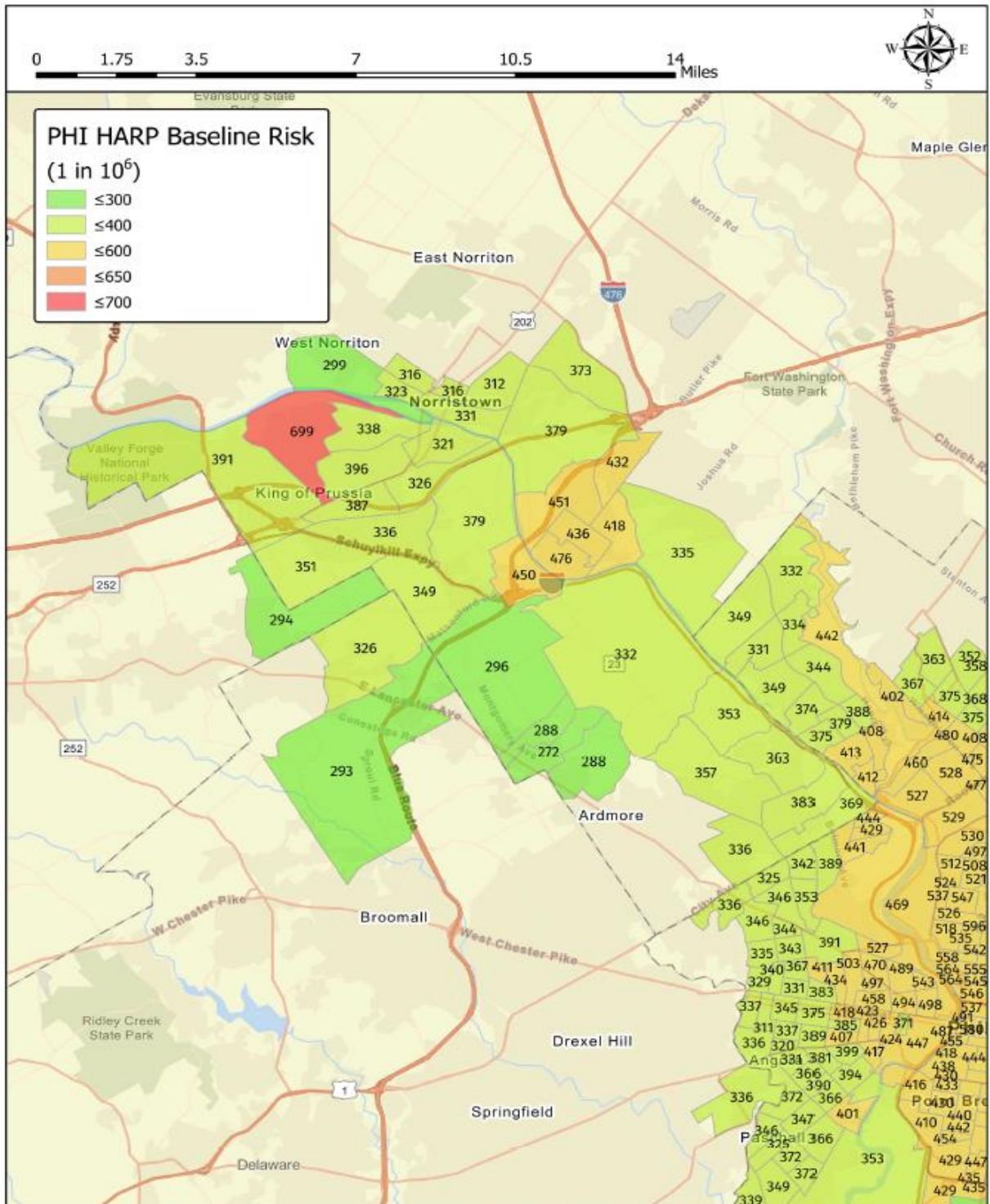


Figure 6-124. N. Philadelphia Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the Philadelphia community is 699.3 cancer cases per million residents for census tract 42091205808, with a population of 5,340 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Philadelphia community is 695 cancer cases expected over a 70-year timeline among a total community population of 1,670,574.

Figure 6-125. S. Philadelphia Reduced NATA/HARP DPM Hybrid Risks

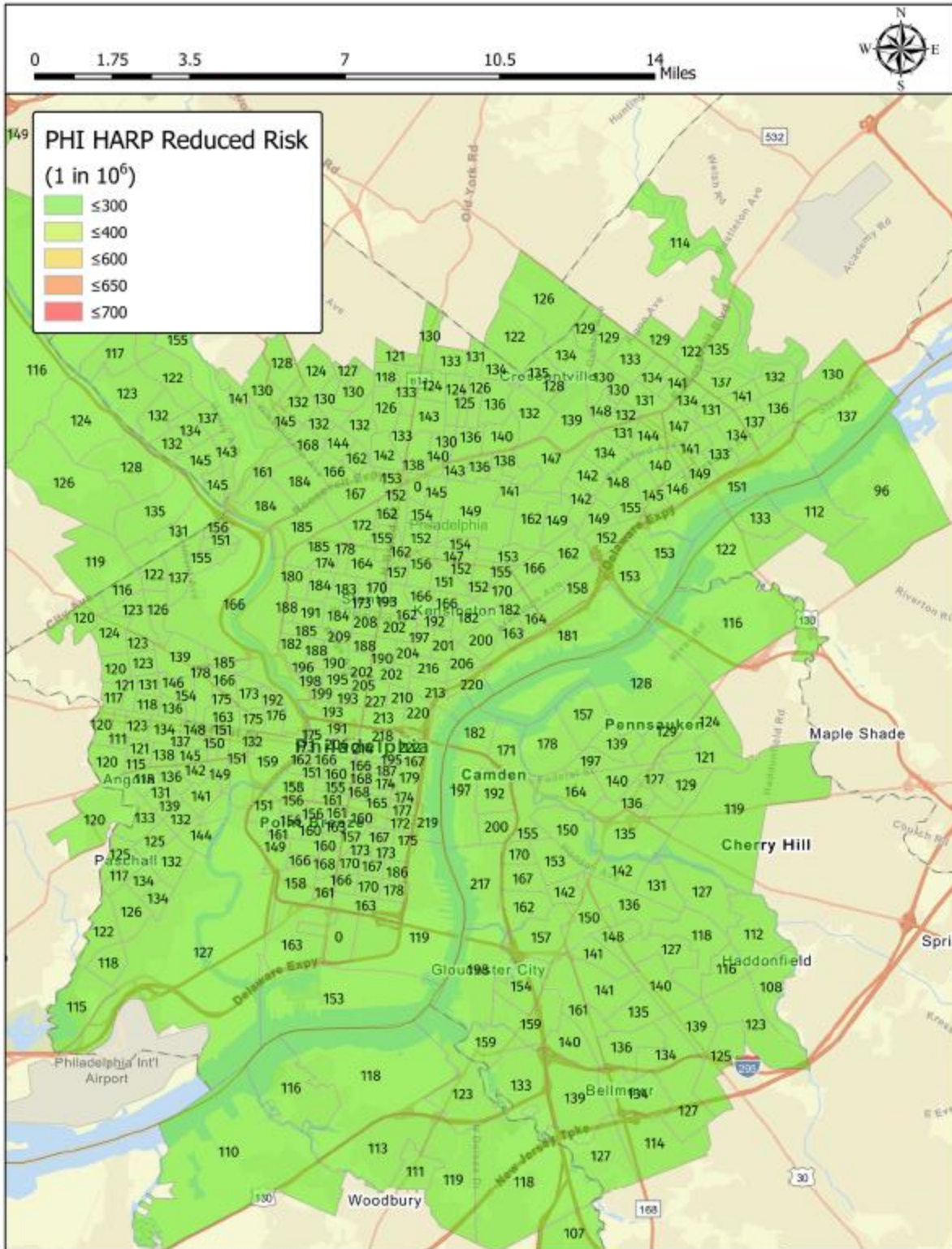
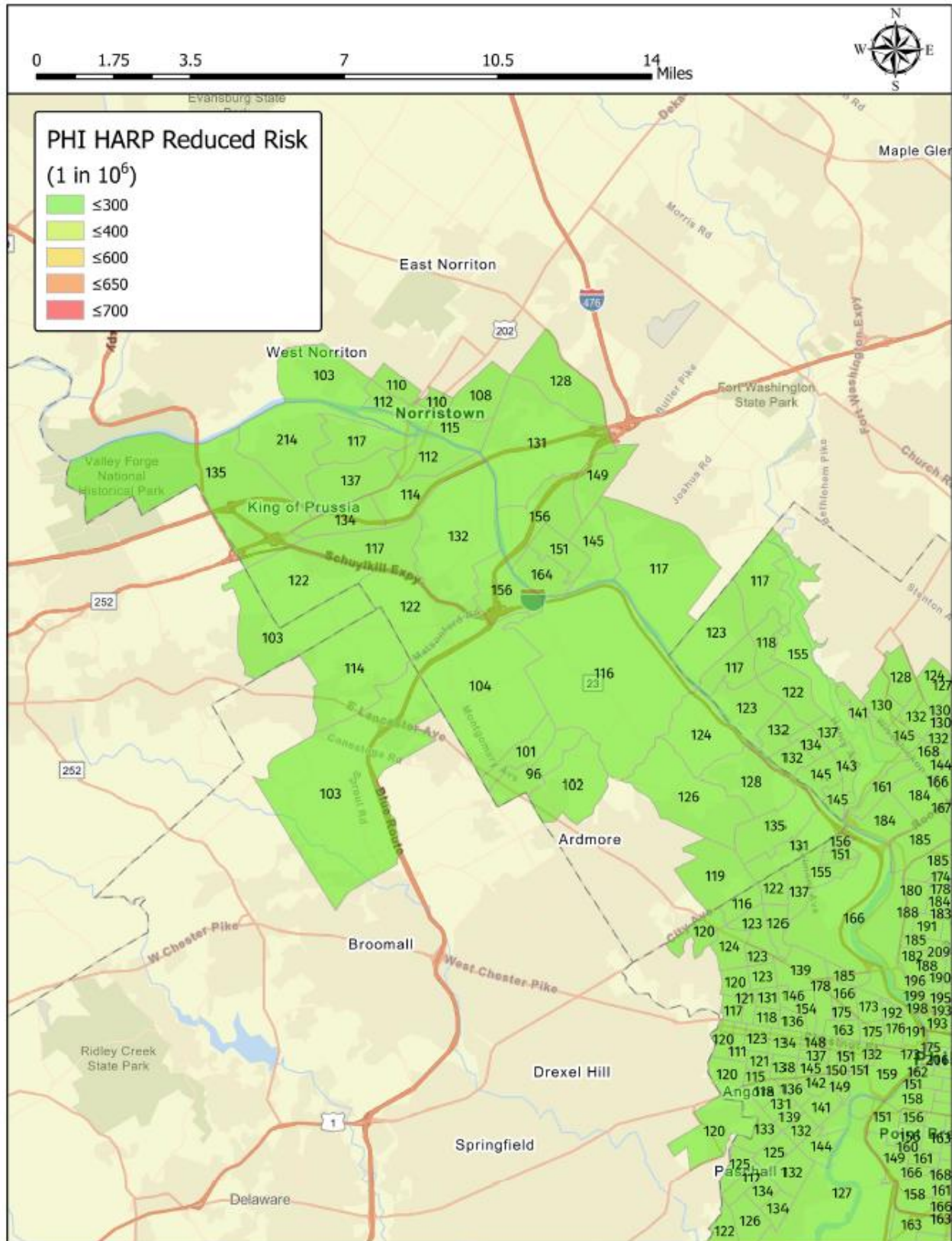


Figure 6-126. N. Philadelphia Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Philadelphia community becomes 227.4 cancer cases per million residents for census tract 42101013200, with a population of 2,914 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Philadelphia community becomes 247 cancer case expected over a 70-year timeline among a total community population of 1,670,574.

6.13.2 Philadelphia Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Philadelphia. The following sources were utilized to generate the HRA.

- Pennsylvania Department of Transportation (PENNDOT) – Traffic Counts (2019 Average Annual Daily Traffic)³⁰
- New Jersey Department of Transportation (NJDOT) – Traffic Counts (2019 Average Annual Daily Traffic)³¹

The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-28.

Table 6-28. Philadelphia-Reading-Camden Source Groups and Emission Rates

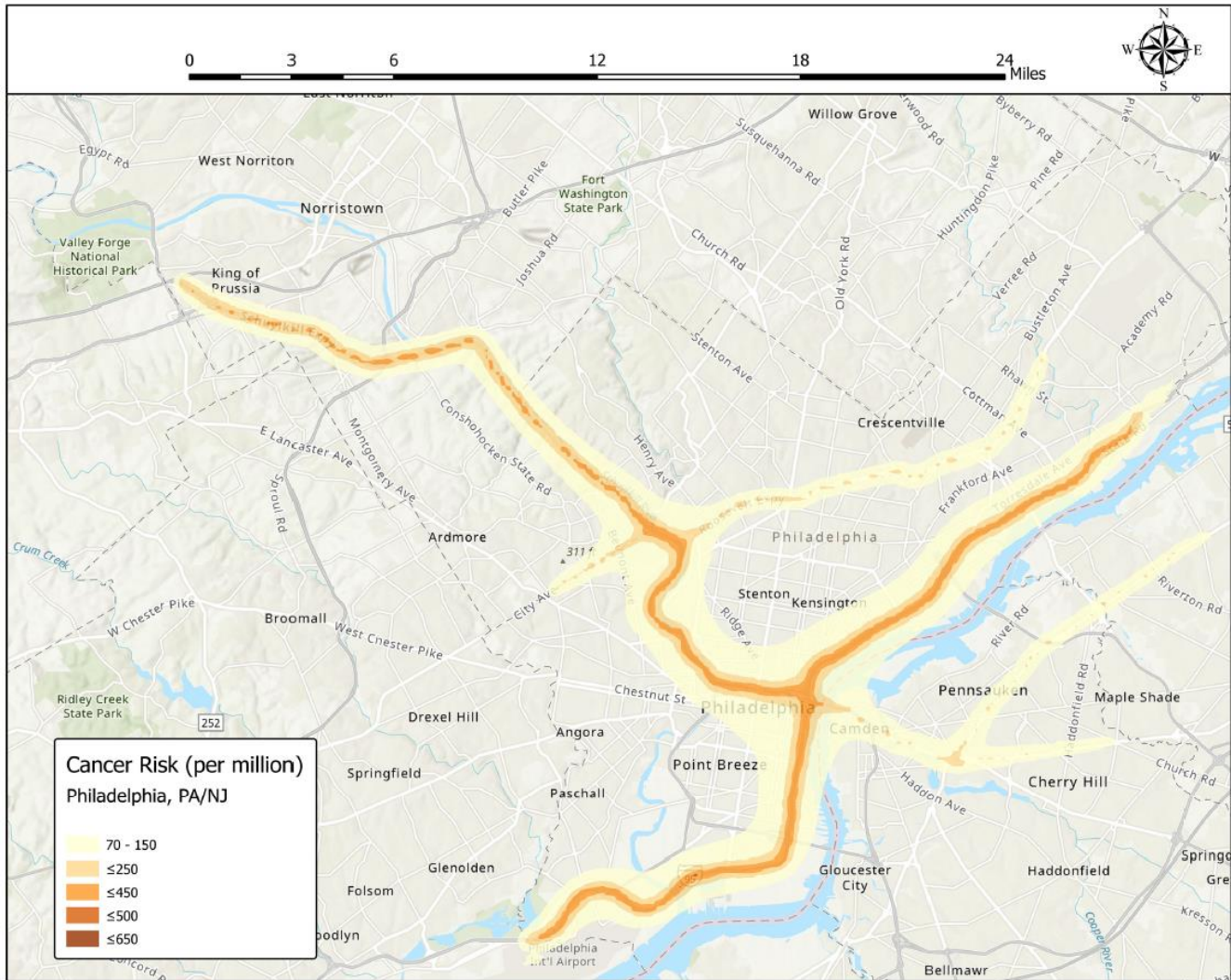
Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
95	State Route 95 – 130,429 AADT	27,132	59.7%
76	State Route 76 – 131,364 AADT	26,272	59.7%
1	State Route 1 – 53,750 AADT	7,357	59.7%
38	State Route 38 – 50,552 AADT	3,682	59.7%
130	State Route 130 – 50,279 AADT	3,936	59.7%

These sources were modeled with unit emission rates in AERMOD, and the Table 6-11. listed emission rates **were input into CARB's HARP software to** determine cancer risks from the DPM concentrations determined by AERMOD. While dispersion characteristics remained the same between baseline and reduced modeling scenarios, **emission rates were reduced according to the number of "old technology" engines combusting diesel, based on source type. The table above shows the Proportion of "Old Technology" Engine Emissions** where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

³⁰ https://gis.penndot.gov/BPR_PDF_FILES/MAPS/Traffic/Traffic_Volume/Historical_Statewide_TVM_Maps/Statewide_2019.pdf

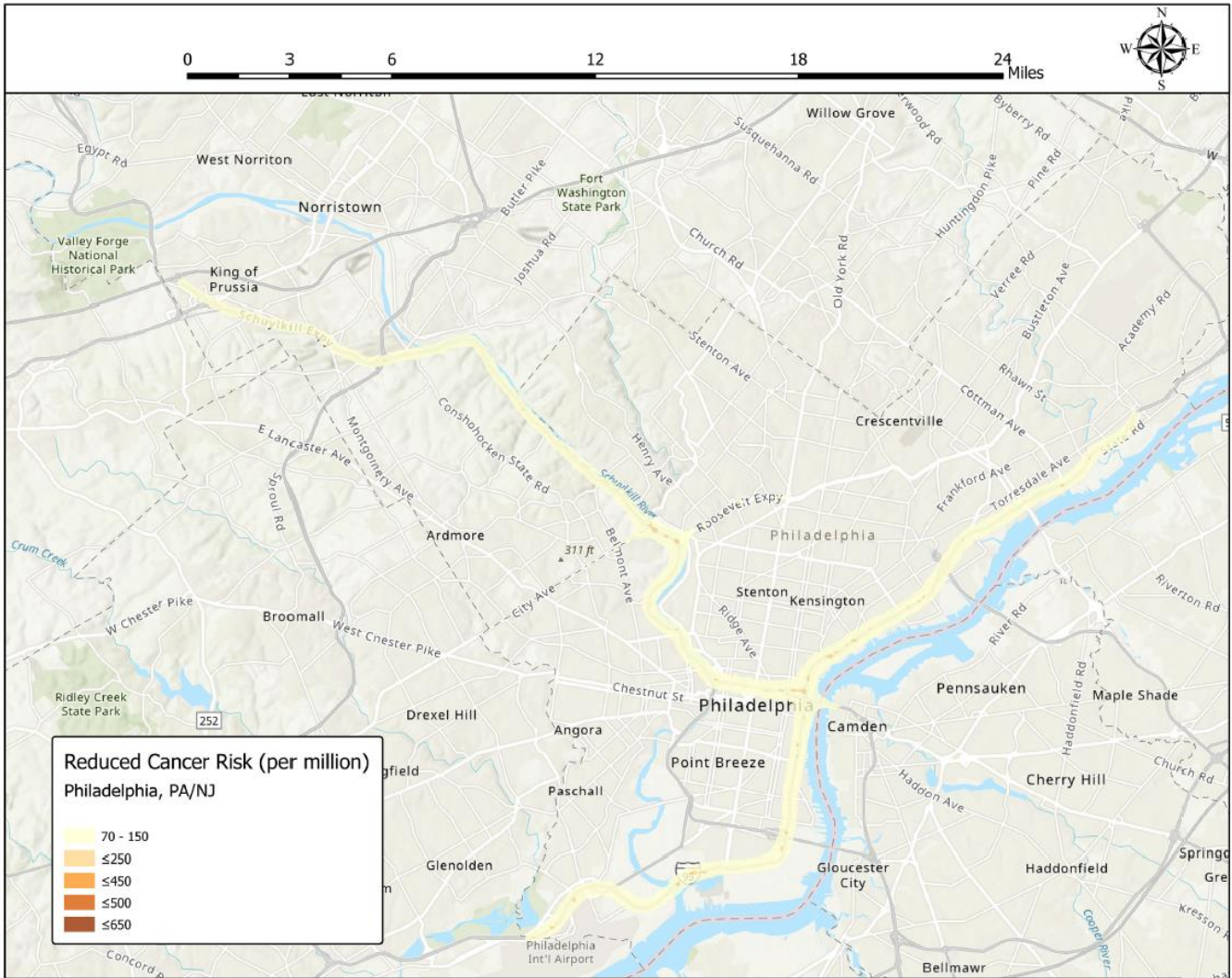
³¹ <https://www.njtms.org/map/>

Figure 6-127. Philadelphia Baseline Health Risk Assessment Isoleths



The site-specific HRA shows that the point of maximum impact (PMI) is similar to the NATA/HARP evaluation, with an impact of 617 cancer cases per million residents. This PMI does not occur at a residential receptor, though, and does not represent an actual risk to residences in the area. The MEIR occurs at 492,272 m E, and 4,426,391.7 m N (NAD 83, UTM Zone 18), with a baseline risk of 535 cancer cases per million residents. This MEIR is higher than the NATA/HARP hybrid risks evaluated for that census tract (42101037800) with a total risk of 507 in a million. This HRA does not capture all of the cancer-causing sources in the area but does demonstrate that NATA values are in-line with the site-specific demonstration with some extremely high local maxima due to local residences proximity to highways.

Figure 6-128. Philadelphia Reduced Health Risk Assessment Isoleths



The reduced cancer risk PMI and MEIR are 382 and 216 in 1 million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 319 in 1 million at the MEIR.

6.13.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-29 below.

Table 6-29. Philadelphia Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction - Nonfatal	123.0	\$3,927,909
Asthma Symptoms - Albuterol use	14,603	\$5,047
ER visits - All Cardiac Outcomes	12.3	\$14,266
ER visits - respiratory	29.6	\$25,850
HA - All Respiratory	3.5	\$65,193
HA - Alzheimers Disease	15.2	\$186,337
HA - Cardio- Cerebro- and Peripheral Vascular Disease	5.7	\$89,280
HA - Parkinsons Disease	3.2	\$40,893
HA - Respiratory-2	0.9	\$0
HA - Respiratory-2 HA All Respiratory	4.3	\$0
Incidence - Asthma	110.2	\$4,921,398
Incidence - Hay Fever/Rhinitis	703.4	\$421,976
Incidence - Lung Cancer	5.8	\$72,704
Incidence - Out of Hospital Cardiac Arrest	0.7	\$23,786
Incidence - Stroke	2.4	\$80,143
Minor Restricted Activity Days	33,714	\$2,345,859
Mortality - All Cause	45.2	\$353,061,728
Work Loss Days	5,717	\$1,121,651
Total		\$366,404,019

6.14 Houston, Texas

6.14.1 NATA Health Risks

The subsections below review the NATA data available for the Houston, TX (Houston) community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-129 shows the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

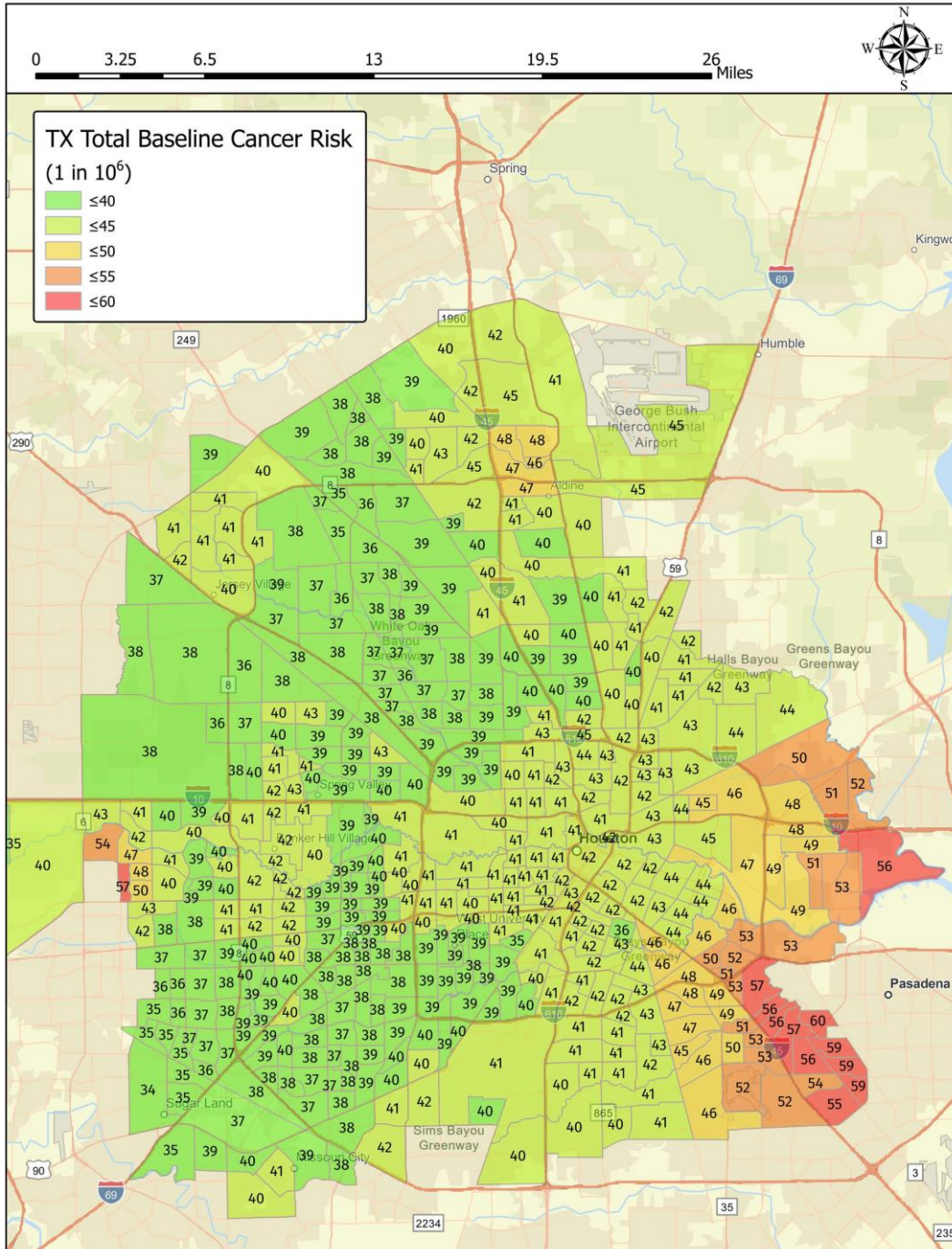
Figure 6-130 shows those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-131 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Houston community.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the Houston community.

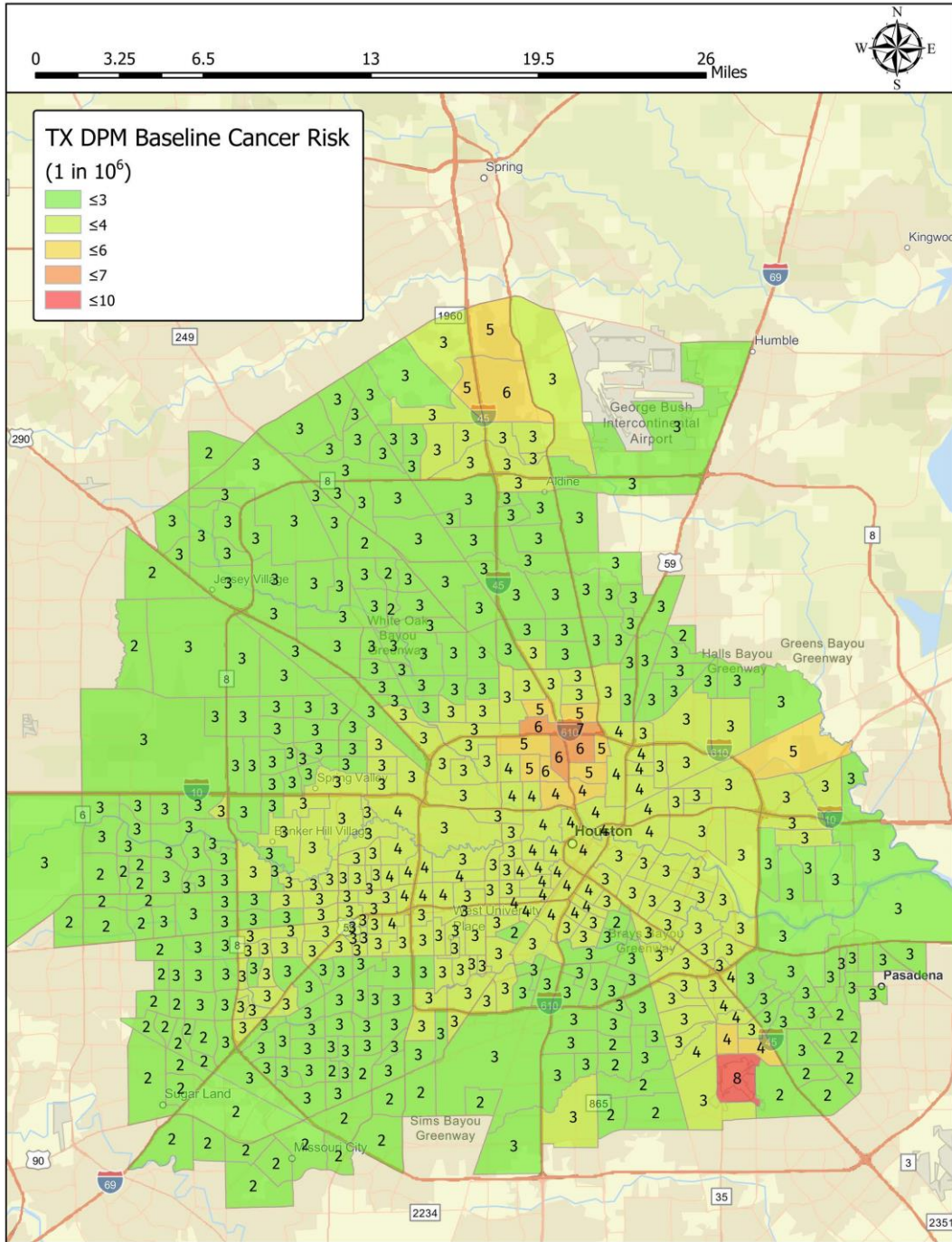
6.14.1.1 NATA Risk Data

Figure 6-129. Houston Baseline NATA Total Cancer Risks



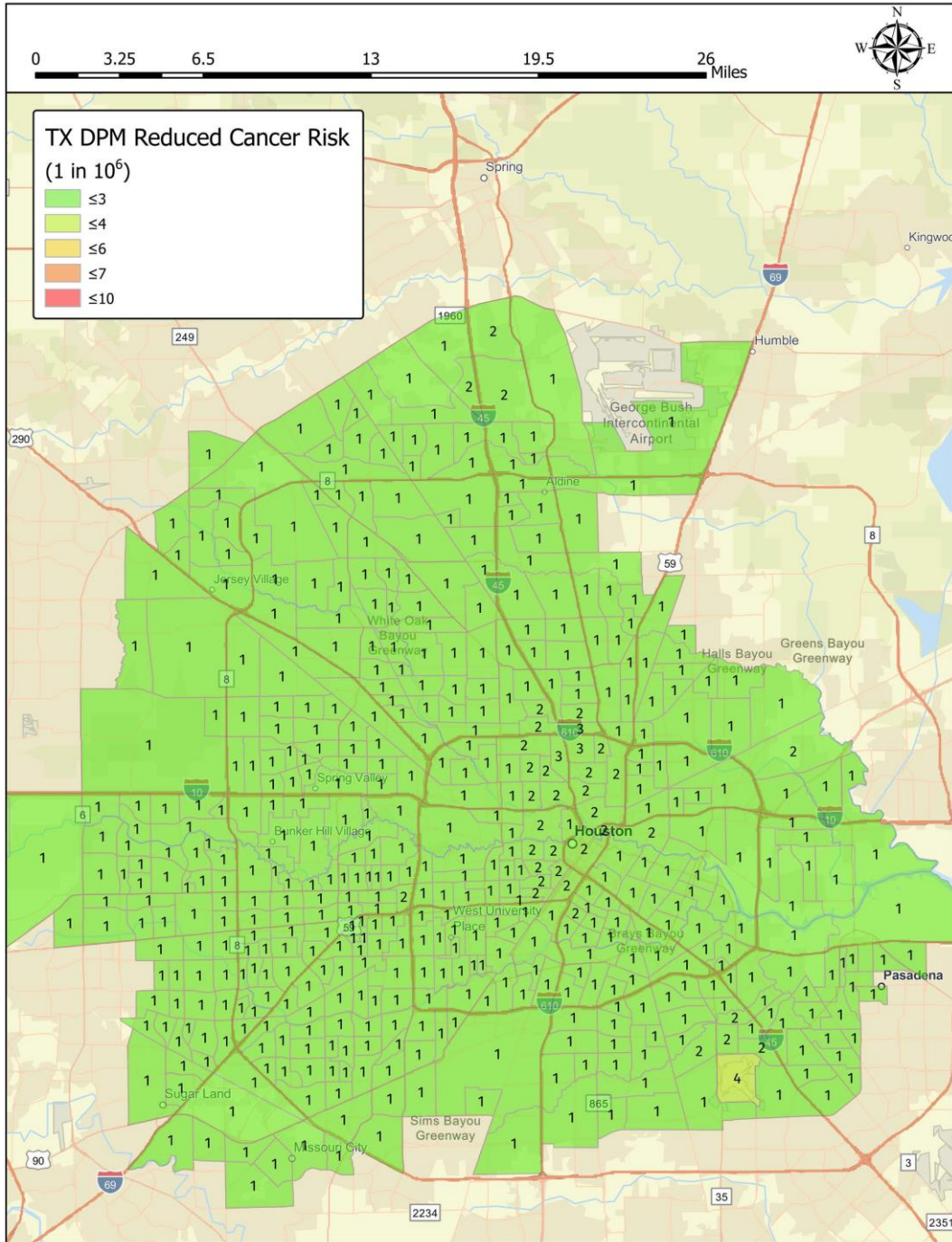
According to the NATA, the maximum baseline cancer risk in the Houston community is 224 cancer cases per million residents for census tract 48201451700, with a population of 3,407 residents. When accounting for all of the communities assessed, the total cancer burden for the Houston community is 96 cancer cases expected over a 70-year timeline among a total community population of 2,298,349.

Figure 6-130. Houston Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the Houston community is 8 cancer cases per million residents for census tract 48201980000, with a population of 33 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Houston community is 7 cancer cases expected over a 70-year timeline among a total community population of 2,298,349.

Figure 6-131. Houston Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Houston community becomes 4 cancer cases per million residents for census tract 48201980000, with a population of 33 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Houston community becomes 3 cancer cases expected over a 70-year timeline among a total community population of 2,298,349.

6.14.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB's HARP program with OEHHA cancer unit risk values. The data is outlined in the following order:**

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program.**

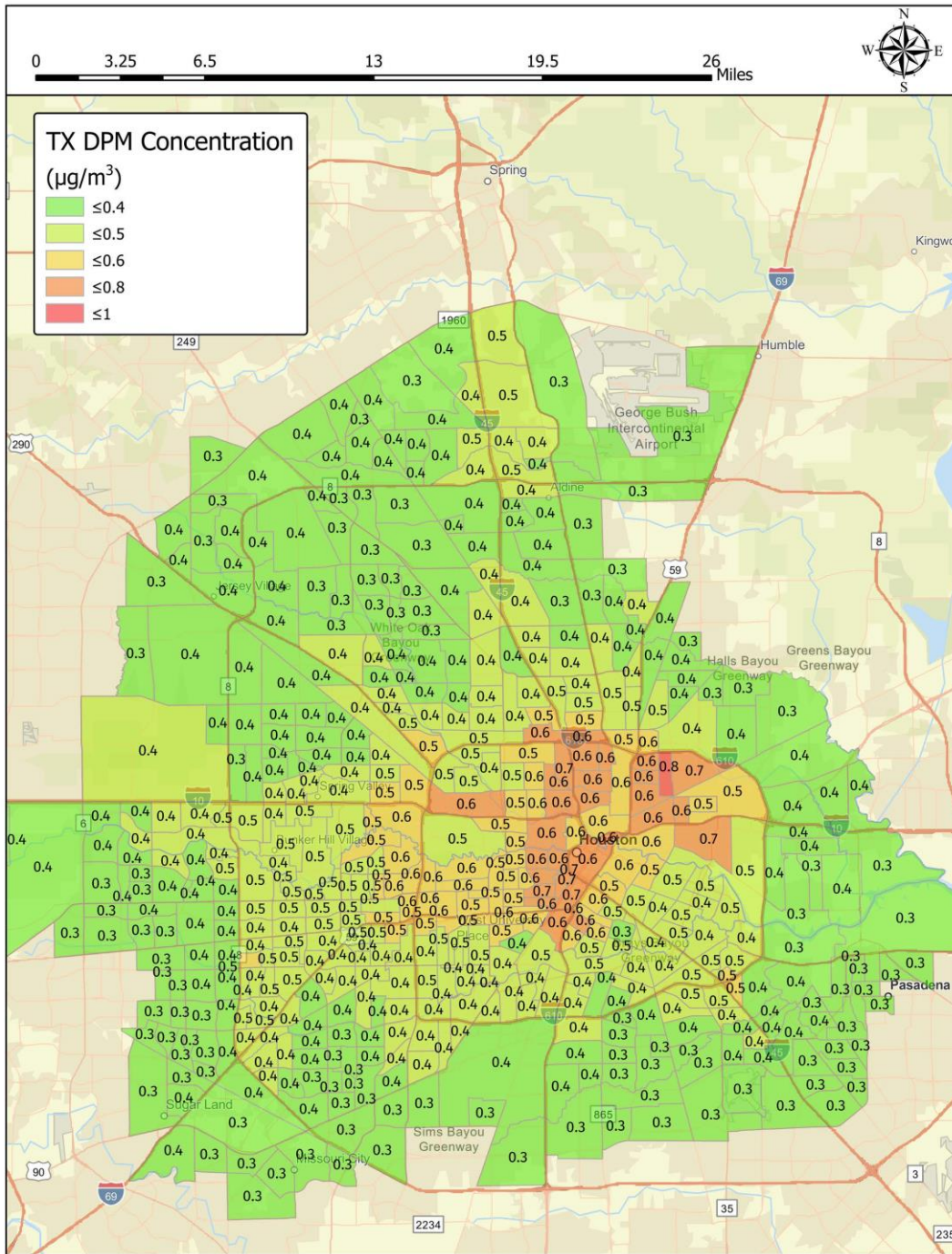
Figure 6-132 shows the baseline DPM concentrations provided by the NATA.

Figure 6-133 shows the baseline DPM-specific cancer risks as determined using the NATA concentration **values and CARB's HARP program.**

Figure 6-134 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the Houston community.

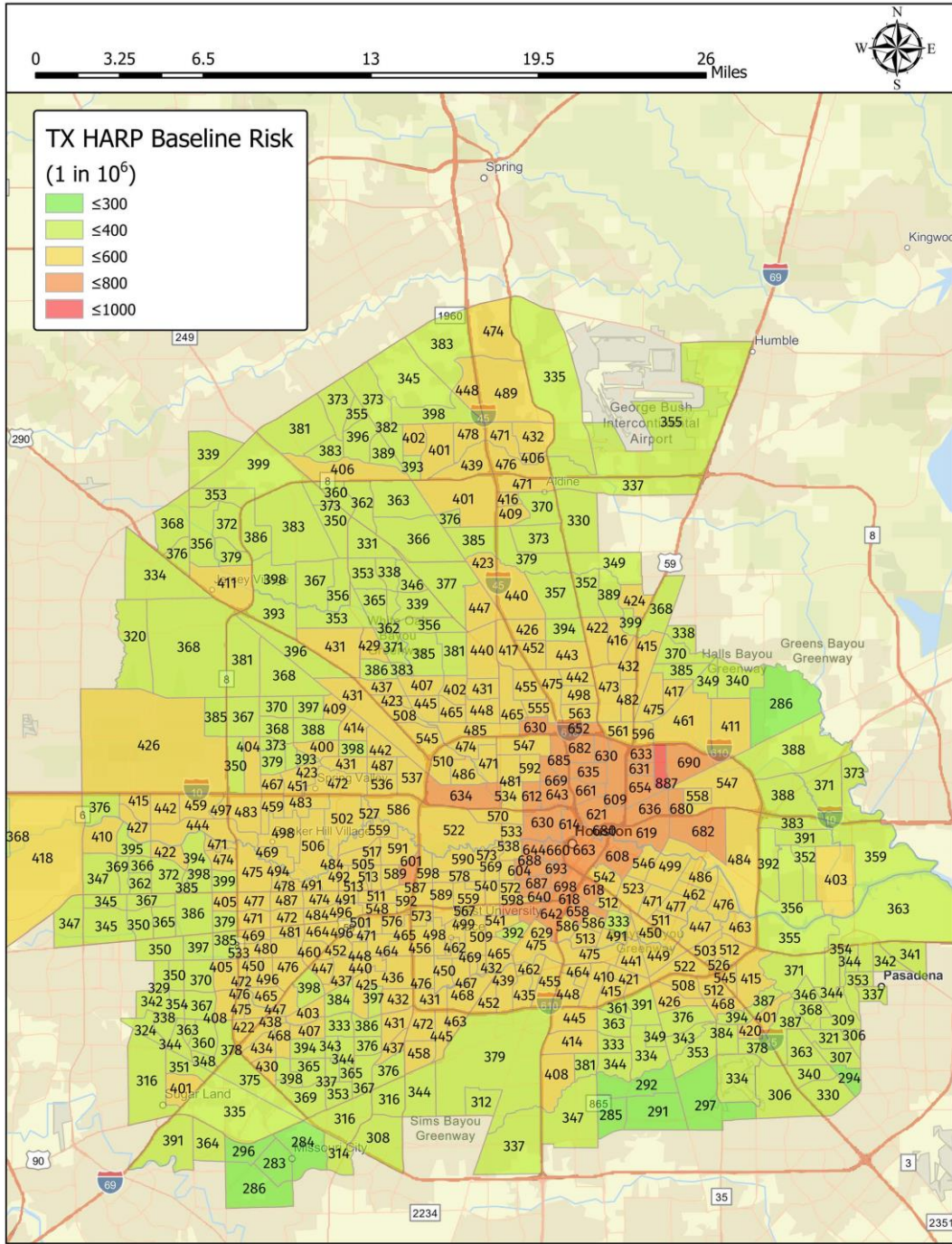
Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the Houston community.

Figure 6-132. Houston Baseline NATA DPM Concentrations



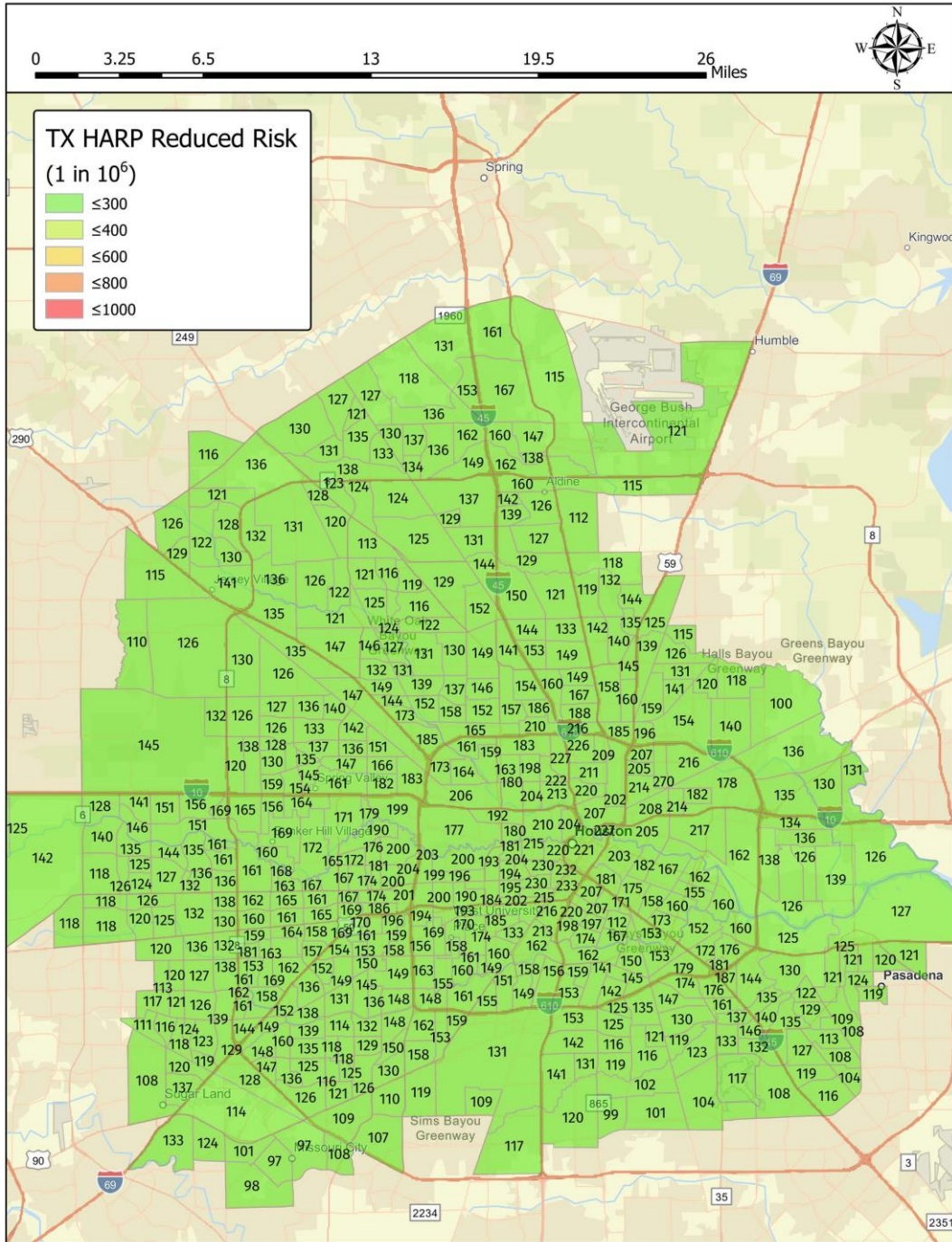
According to the NATA, the maximum baseline DPM concentration in the Houston community is 0.85 $\mu\text{g}/\text{m}^3$ for census tract 48201211200, with a population of 2,831 residents. The average DPM concentration of the Houston community is 0.42 $\mu\text{g}/\text{m}^3$.

Figure 6-133. Houston Baseline NATA/HARP DPM Hybrid Risks



Using NATA DPM concentrations and OEHHA cancer unit risk values, the maximum DPM-specific baseline cancer risk in the Houston community is 887 cancer cases per million residents for census tract 48201211200, with a population of 2,831 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the Houston community is 998 cancer cases expected over a 70-year timeline among a total community population of 2,298,349.

Figure 6-134. Houston Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the Houston community becomes 270 cancer cases per million residents for census tract 48201211200, with a population of 2,831 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the Houston community becomes 340 cancer cases expected over a 70-year timeline among a total community population of 2,298,349.

6.14.2 Houston Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for Houston. The following sources were utilized to generate the HRA.

- Texas Department of Transportation (TXDOT) – Traffic Counts (2020 Average Annual Daily Traffic)³²

The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-30.

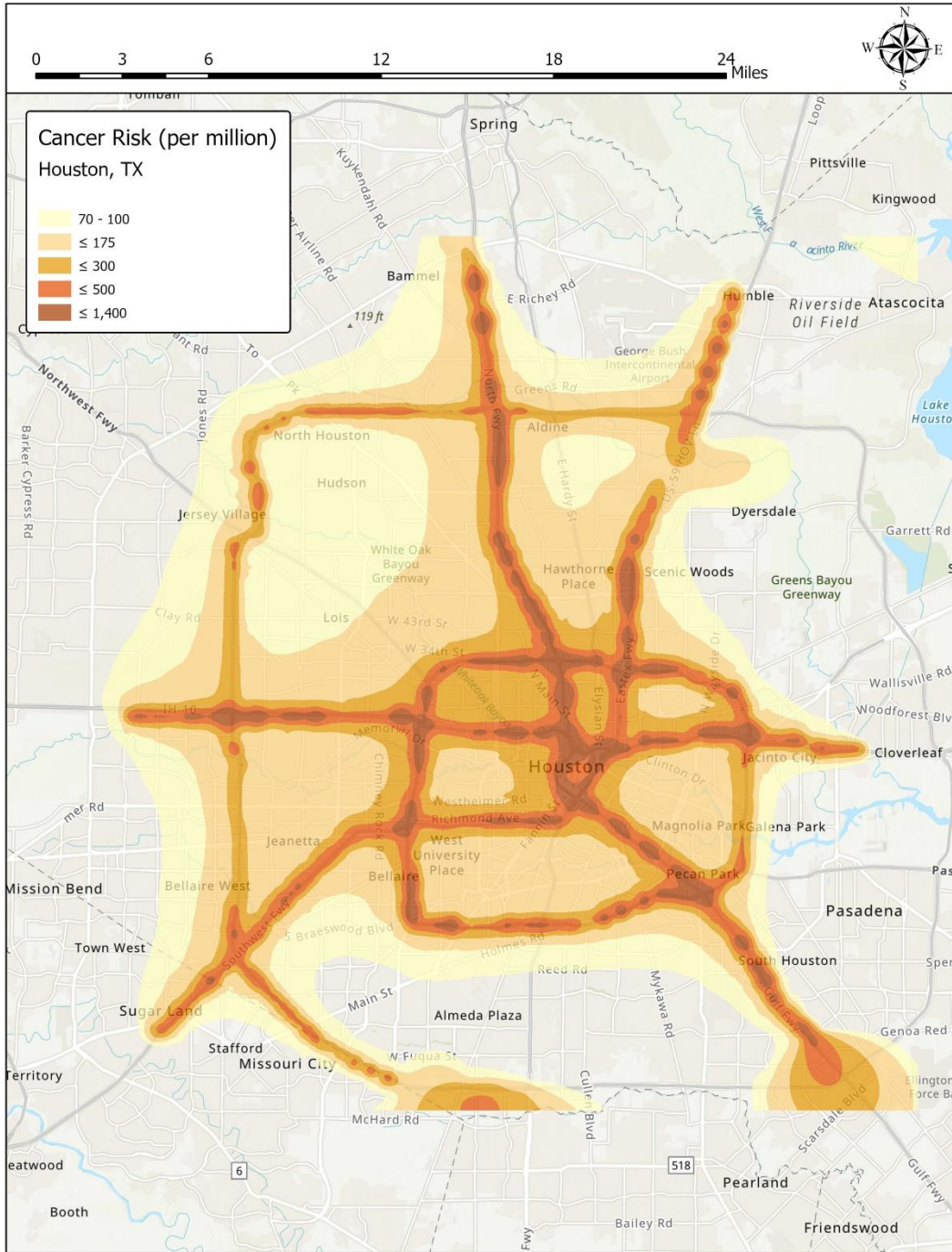
Table 6-30. Houston Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
I-10	I-10 – 180,469 AADT	48,070	59.7%
I-610	I-610 – 162,907 AADT	61,774	59.7%
TX8	Texas Loop 8 – 93,914 AADT	44,033	59.7%
I-45	I-45 – 196,594 AADT	63,484	59.7%
I-69	I-69 – 187,536 AADT	70,084	59.7%

These sources were modeled with unit emission rates in AERMOD, and the Table 6-30 listed emission rates were input into CARB's HARP software to determine cancer risks from the DPM concentrations determined by AERMOD. While dispersion characteristics remained the same between baseline and reduced modeling scenarios, emission rates were reduced according to the number of "old technology" engines combusting diesel, based on source type. The table above shows the Proportion of "Old Technology" Engine Emissions where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

³² <https://txdot.maps.arcgis.com/apps/webappviewer/index.html?id=06fea0307dda42c1976194bf5a98b3a1>

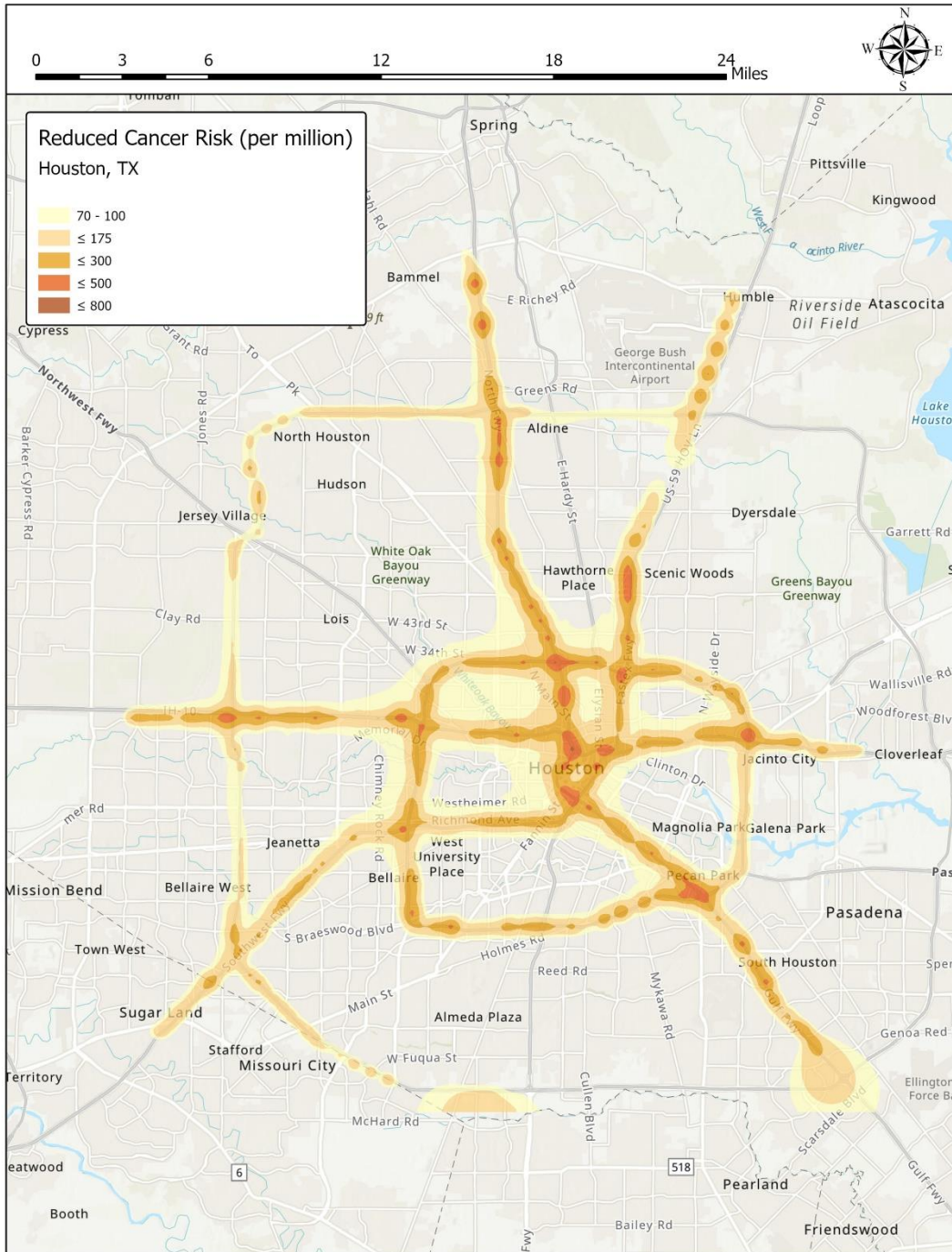
Figure 6-135. Houston Baseline Health Risk Assessment Isopeleths



The site-specific HRA shows that the point of maximum impact (PMI) is substantially higher than the NATA/HARP evaluation, with an impact of 1,446 cancer cases per million residents. This PMI does not occur at a residential receptor, though, and does not represent an actual risk to residences in the area. The MEIR occurs at 273,929.1 m E, and 3,299,642.1 m N (NAD 83, UTM Zone 15), with a baseline risk of 1,124 cancer cases per million residents. This MEIR is higher than the NATA/HARP hybrid risks evaluated for that census tract (48201210800) with a total risk of 609 in a million. This HRA does not capture all of the cancer-causing

sources in the area but does demonstrate that NATA values are in-line with the site-specific demonstration with some extremely high local maxima due to local residences proximity to highways.

Figure 6-136. Houston Reduced Health Risk Assessment Isopleths



The reduced cancer risk PMI and MEIR are 583 and 453 in 1 million, respectively, both in the same locations as the baseline risk plots. This represents a risk reduction of 671 in 1 million at the MEIR.

6.14.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using USEPA's BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-31 below.

Table 6-31. Houston Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	124.8	\$4,241,890
Asthma Symptoms - Albuterol use	24,044	\$8,309
ER visits - All Cardiac Outcomes	16.0	\$18,548
ER visits – Respiratory	44.2	\$38,629
HA – All – Respiratory	4.9	\$76,093
HA – Alzheimer's Disease	8.9	\$107,101
HA – Cardio Cerebro- and Peripheral Vascular Disease	4.9	\$75,752
HA – Parkinson's Disease	2.1	\$27,603
HA – Respiratory-2	0.8	\$0
HA – Respiratory-2 HA – All Respiratory	5.7	\$0
Incidence – Asthma	186.6	\$8,331,216
Incidence – Hay Fever/Rhinitis	1,155	\$692,949
Incidence – Lung Cancer	6.0	\$76,390
Incidence – Out of Hospital Cardiac Arrest	0.8	\$27,758
Incidence – Stroke	2.3	\$78,445
Minor Restricted Activity Days	47,822	\$3,327,529
Mortality – All Cause	36.6	\$283,781,827
Work Loss Days	8,184	\$1,413,645
Total		\$302,323,685

6.15 St. Louis, Missouri

6.15.1 NATA Health Risks

The subsections below review the NATA data available for the St. Louis, MO (St. Louis) community. The data is outlined in the following order:

- Baseline NATA Total Cancer Risks
- Baseline NATA DPM Cancer Risks
- Reduced NATA DPM Cancer Risks

As stated previously, NATA indirectly determines DPM cancer risk by utilizing the individual exhaust component emission rates and toxicity factors. The census tract DPM concentrations provided by NATA are not utilized to determine cancer risks in the NATA evaluation. Therefore, census tract DPM concentrations are not shown in this section, and the NATA-specific review only utilizes NATA raw data to determine the health risk reductions due to a change to biodiesel.

Figure 6-137 shows the Baseline NATA Total Cancer Risk. This total cancer risk encompasses all sources in the area.

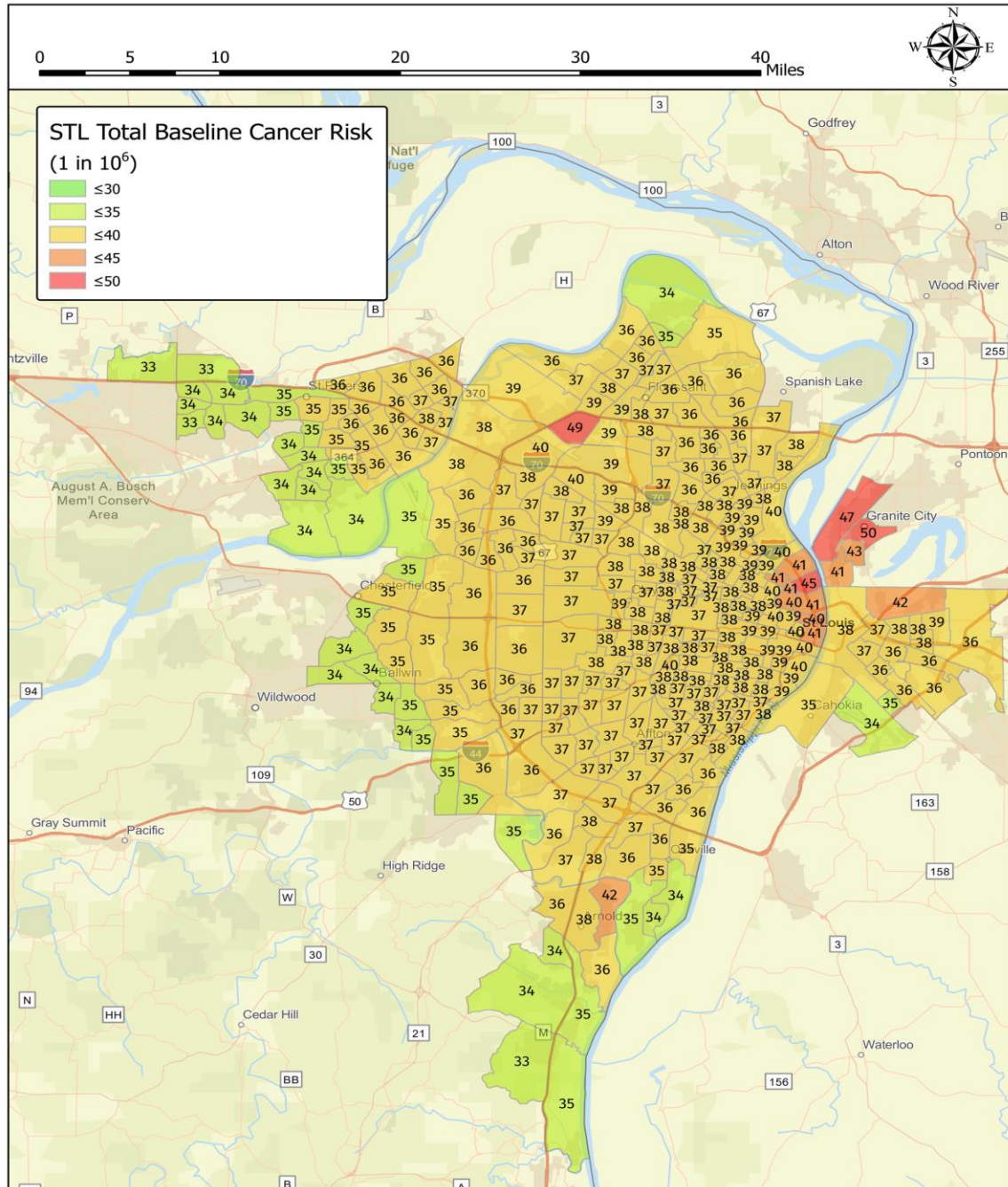
Figure 6-138 shows those cancer risks specific to DPM emissions as determined using NATA raw data.

Figure 6-139 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the St. Louis community.

Because the NATA analysis utilized EPA-specific health risk values, the baseline and reduced cancer risks will be orders of magnitude lower than any equivalent analysis using OEHHA risk values. Therefore, the results of this analysis can be considered the low-end estimate of baseline and reduced cancer risks in the St. Louis community.

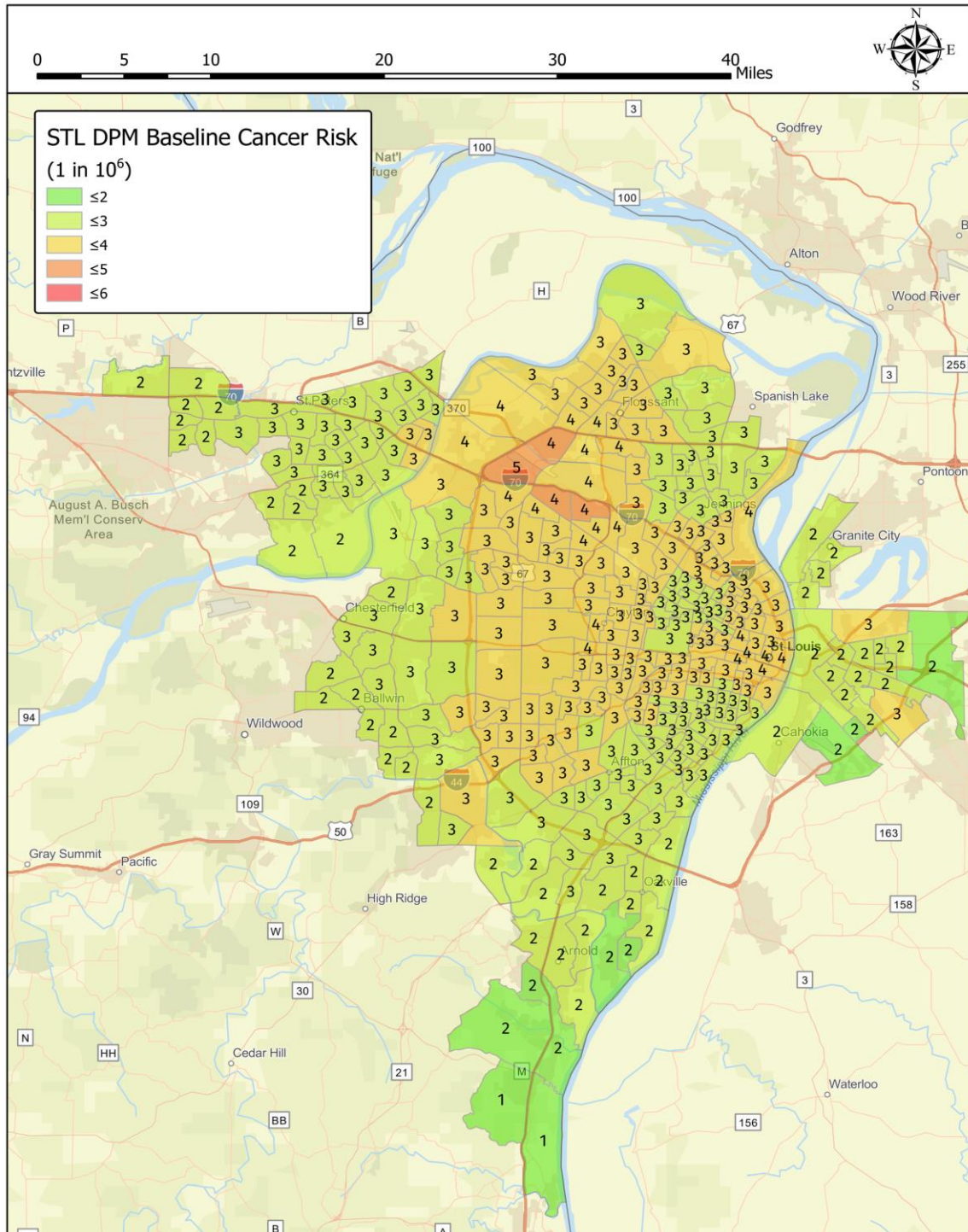
6.15.1.1 NATA Risk Data

Figure 6-137. St. Louis Baseline NATA Total Cancer Risks



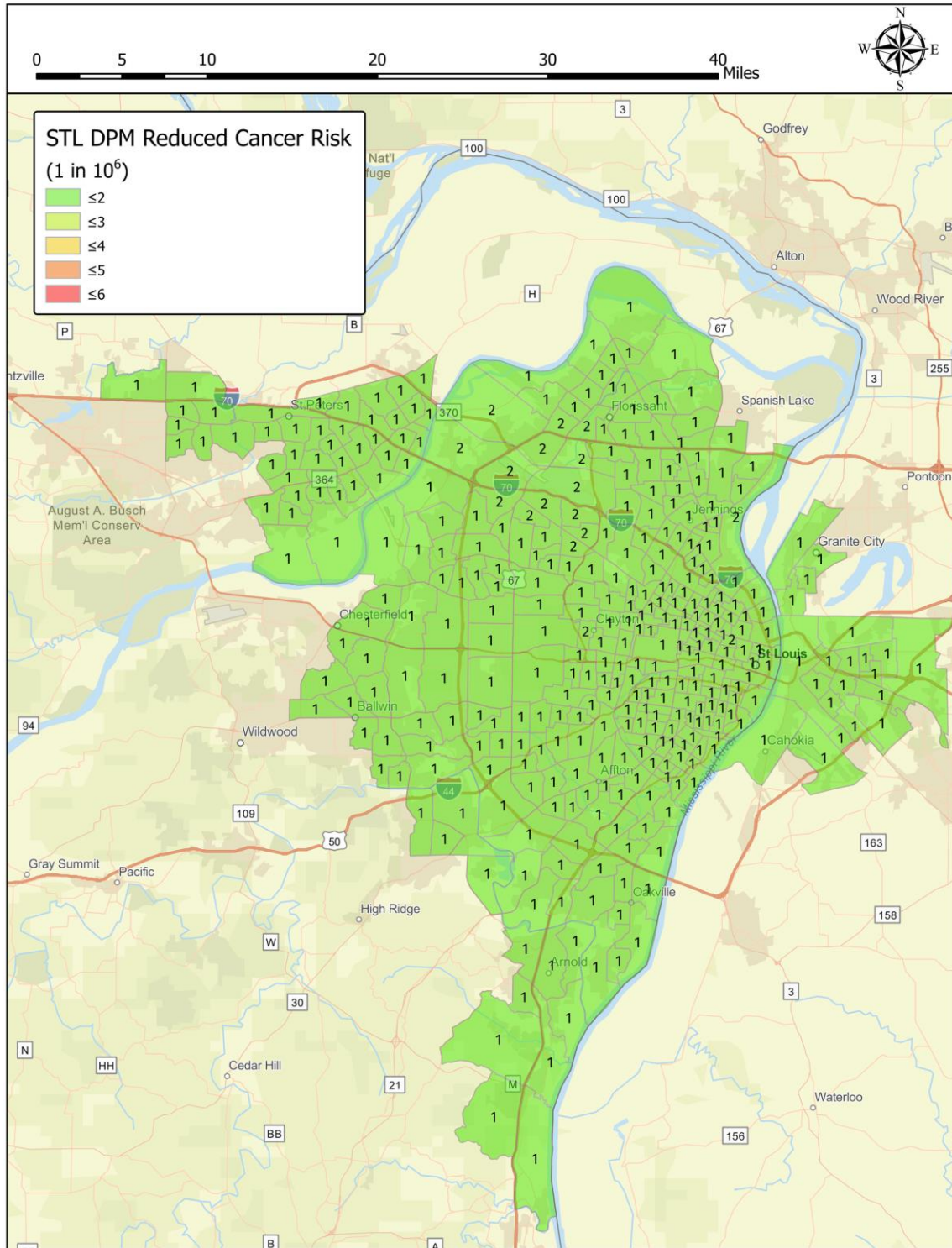
According to the NATA, the maximum baseline cancer risk in the St. Louis community is 50 cancer cases per million residents for census tract 17119404000, with a population of 2,785 residents. When accounting for all of the communities assessed, the total cancer burden for the St. Louis community is 56 cancer cases expected over a 70-year timeline among a total community population of 1,518,290.

Figure 6-138. St. Louis Baseline NATA DPM Cancer Risks



According to the NATA, the maximum DPM-specific baseline cancer risk in the St. Louis community is 5 cancer cases per million residents for census tract 29189213101, with a population of 4,606 residents. When accounting for all of the communities assessed, the baseline DPM-specific cancer burden for the St. Louis community is 4 cancer cases expected over a 70-year timeline among a total community population of 1,518,290.

Figure 6-139. St. Louis Reduced NATA DPM Cancer Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the St. Louis community becomes 2 cancer cases per million residents for census tract 29189213101, with a population of 4,606 residents. When accounting for all of the communities assessed,

the reduced DPM-specific cancer burden for the St. Louis community becomes 2 cancer cases expected over a 70-year timeline among a total community population of 1,518,290.

6.15.1.2 NATA Data with HARP Risk Factors

The subsections below utilize the DPM concentration values provided by the NATA, which are then **evaluated using CARB's HARP program with OEHHA cancer unit risk values. The data is outlined in the following order:**

- Baseline NATA DPM Concentrations
- Baseline NATA/HARP DPM Hybrid Risks
- Reduced NATA/HARP DPM Hybrid Risks

As stated previously, OEHHA cancer unit risk values can be orders of magnitude higher than EPA risk values. The census tract DPM concentrations provided by NATA were therefore utilized to determine cancer risks in combination with OEHHA cancer unit risk values. The NATA DPM concentrations are shown, along with **baseline and reduced cancer risks using CARB's HARP program.**

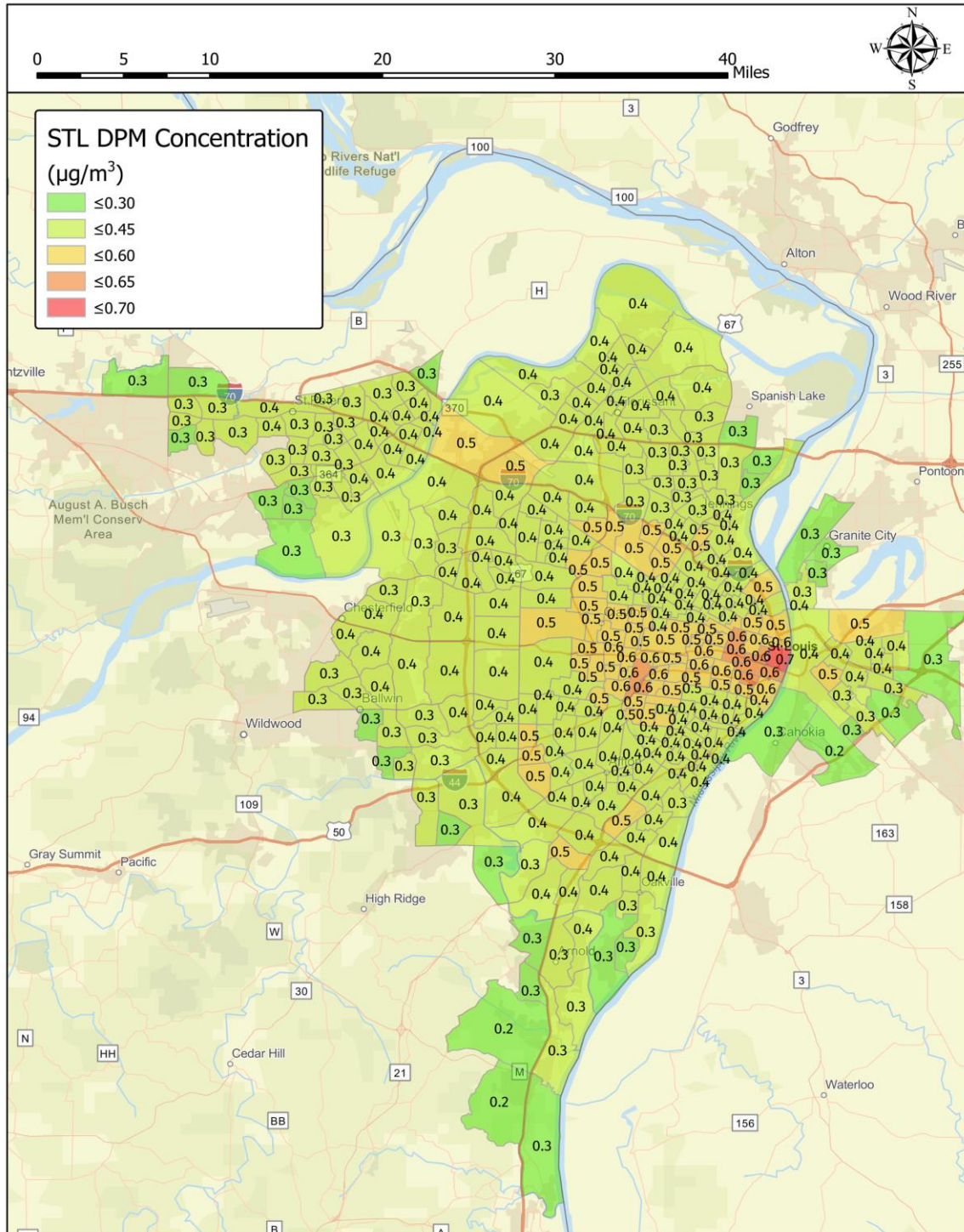
Figure 6-140 shows the baseline DPM concentrations provided by the NATA.

Figure 6-141 shows the baseline DPM-specific cancer risks as determined using the NATA concentration **values and CARB's HARP program.**

Figure 6-142 shows the reduced cancer risks specific to DPM emissions assuming a 100% change to biodiesel fuel for all emissions sources in the St. Louis community.

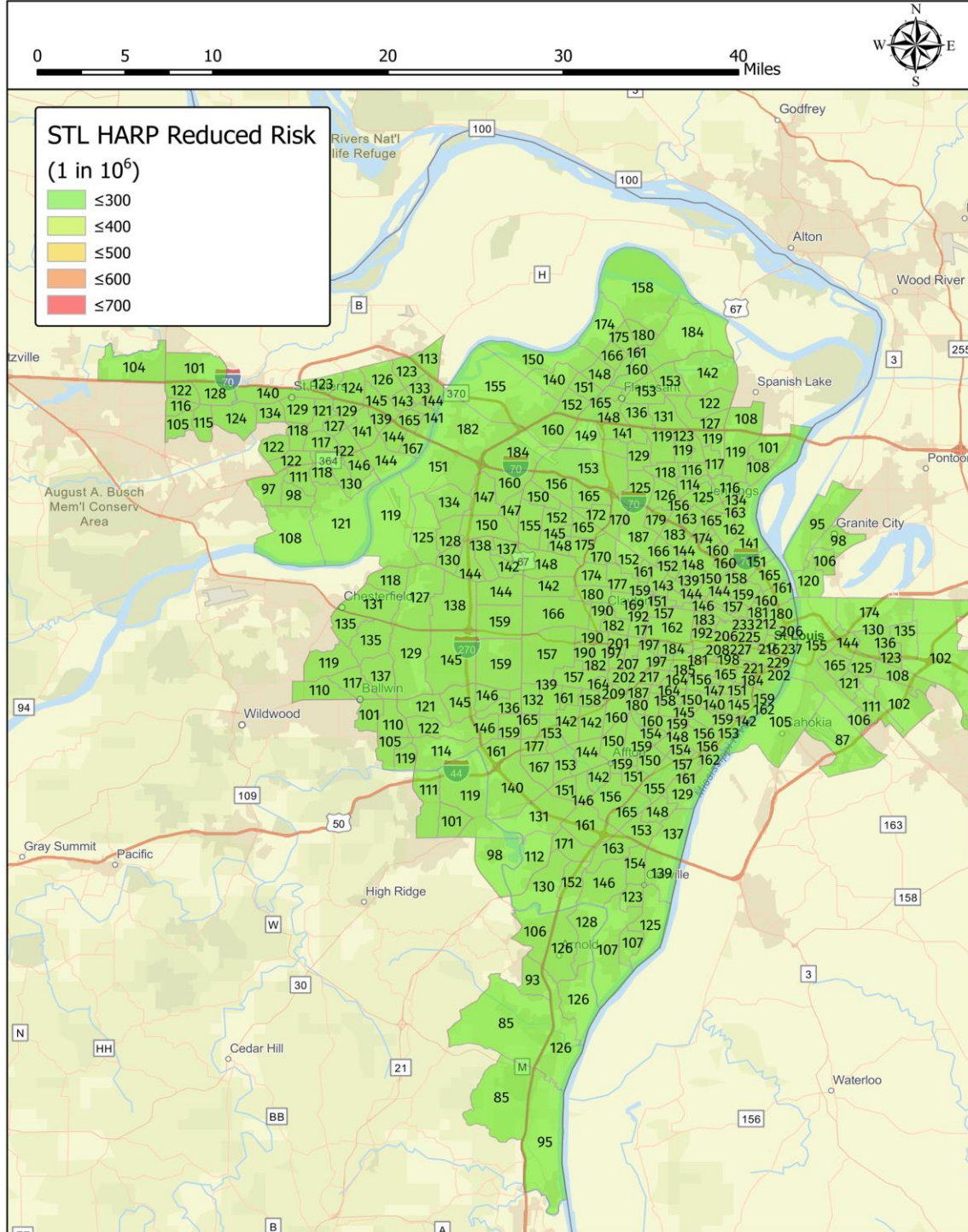
Because this hybrid NATA/HARP analysis utilized OEHHA specific health risk values, the baseline and reduced cancer risks are orders of magnitude higher than an equivalent analysis using EPA cancer unit risk values. Therefore, the results of this analysis can be considered the high-end estimate of baseline and reduced cancer risks in the St. Louis community.

Figure 6-140. St. Louis Baseline NATA DPM Concentrations



According to the NATA, the maximum baseline DPM concentration in the St. Louis community is 0.66 $\mu\text{g}/\text{m}^3$ for census tract 29510125600, with a population of 4,113 residents. The average DPM concentration of the St. Louis community is 0.41 $\mu\text{g}/\text{m}^3$.

Figure 6-142. St. Louis Reduced NATA/HARP DPM Hybrid Risks



Applying the biodiesel exhaust reduction factor outlined in Section 4.2, the maximum DPM-specific reduced cancer risk in the St. Louis community becomes 237 cancer cases per million residents for census tract 29510125600, with a population of 4,113 residents. When accounting for all of the communities assessed, the reduced DPM-specific cancer burden for the St. Louis community becomes 221 cancer cases expected over a 70-year timeline among a total community population of 1,518,290.

6.15.2 St. Louis Site-Specific Health Risk Assessment

While the NATA report is a useful tool for general community assessment of health risks, it should not be utilized to infer findings for specific areas. In order to determine refined health benefits from transition to biodiesel in an existing area of concern, a site-specific HRA was conducted for St. Louis. The following sources were utilized to generate the HRA.

- Missouri Department of Transportation (MODOT) – Traffic Counts (2020 Average Annual Daily Traffic)³³

The emissions sources were modeled with the following source groups in AERMOD, consistent with the report, representing the associated emission rates listed in Table 6-30.

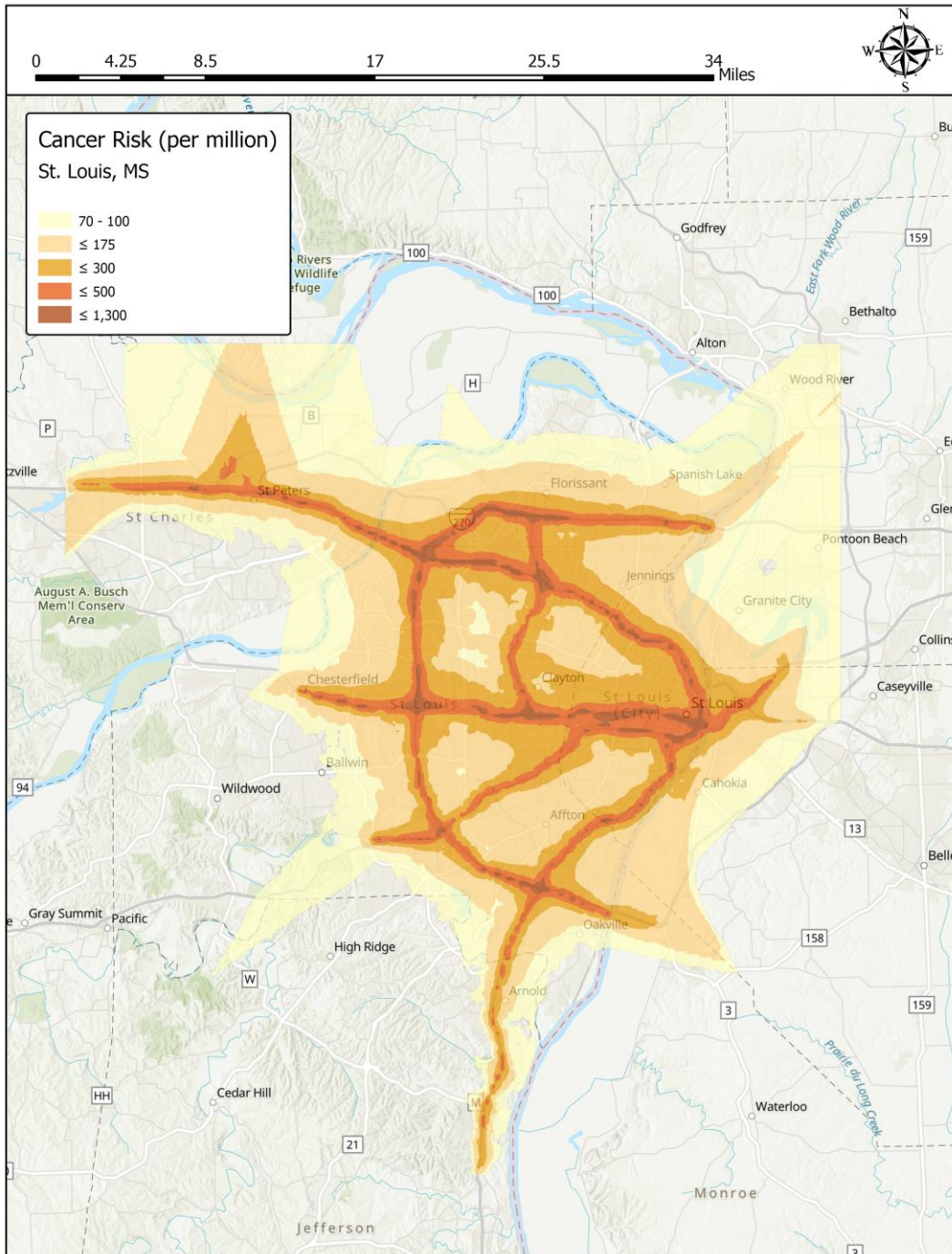
Table 6-32. St. Louis Source Groups and Emission Rates

Source Group	Description	DPM Emissions (lb/yr)	Proportion of "Old Technology" Engine Emissions
I-70	I-70 – 141,125 AADT	49,132	59.7%
I-70 IL	I-70 IL – 24,250 AADT	1,618	59.7%
I-170	I-170 – 107,157 AADT	11,702	59.7%
I-270	I-270 – 148,046 AADT	58,533	59.7%
I-64	I-64 – 146,294 AADT	29,977	59.7%
I-44	I-44 – 102,219 AADT	18,199	59.7%
I-55	I-55 – 113,318 AADT	33,728	59.7%
I-55 IL	I-55 IL – 73,957 AADT	4,322	59.7%

These sources were modeled with unit emission rates in AERMOD, and the Table 6-30 listed emission rates were input into CARB's HARP software to determine cancer risks from the DPM concentrations determined by AERMOD. While dispersion characteristics remained the same between baseline and reduced modeling scenarios, emission rates were reduced according to the number of "old technology" engines combusting diesel, based on source type. The table above shows the Proportion of "Old Technology" Engine Emissions where the DPM reduction factor was taken into account. The subsequent figures show the baseline and reduced cancer risk isopleths from the analysis and include information on the MEIR for the analysis.

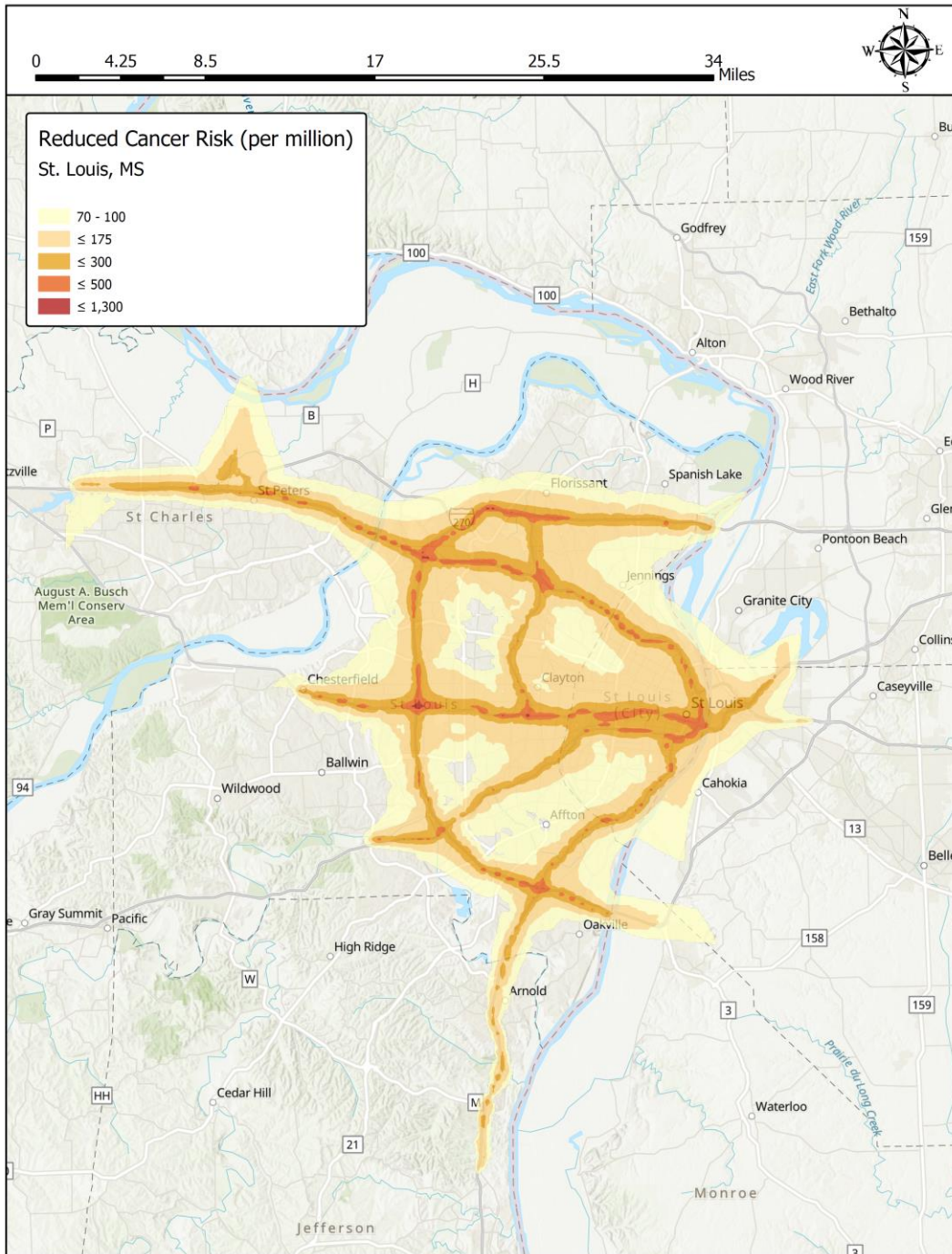
³³ <https://datazoneapps.modot.mo.gov/bi/apps/maps/Home/Index/AADT>

Figure 6-143. St. Louis Baseline Health Risk Assessment Isopleths



The site-specific HRA shows that the point of maximum impact (PMI) is substantially higher than the NATA/HARP evaluation, with an impact of 1,446 cancer cases per million residents. The PMI occurs at a residential receptor located at 271,229.1 m E, and 3,295,592.1 m N (NAD 83, UTM Zone 15) and represents the MEIR. This HRA does not capture all of the cancer-causing sources in the area, but does demonstrate that NATA values are in-line with the site-specific demonstration with some extremely high local maxima due to local residences proximity to highways.

Figure 6-144. St. Louis Reduced Health Risk Assessment Isopleths



The reduced cancer risk PMI and MEIR are 583 in 1 million located in the same locations as the baseline risk plots. This represents a risk reduction of 863 in 1 million.

6.15.3 Valuation of Health Benefits

The health benefits of reduced PM_{2.5} exposure were modeled using **USEPA's** BenMAP model according to the methodology described under Section 4.7. The results are shown in Table 6-31 below.

Table 6-33. St. Louis Valuation of Reduced Incidence Benefits

Endpoint	Reduced Incidence	Benefit Value
Acute Myocardial Infarction Nonfatal	77.8	\$2,529,686
Asthma Symptoms - Albuterol use	8,192	\$2,831
ER visits - All Cardiac Outcomes	7.7	\$8,968
ER visits – Respiratory	16.3	\$14,252
HA – All – Respiratory	2.3	\$43,415
HA – Alzheimer’s Disease	21.6	\$261,411
HA – Cardio Cerebro- and Peripheral Vascular Disease	3.6	\$55,271
HA – Parkinson’s Disease	1.6	\$20,949
HA – Respiratory-2	0.6	\$0
HA – Respiratory-2 HA – All Respiratory	2.8	\$0
Incidence – Asthma	61.5	\$2,744,976
Incidence – Hay Fever/Rhinitis	392.2	\$235,259
Incidence – Lung Cancer	3.3	\$42,378
Incidence – Out of Hospital Cardiac Arrest	0.4	\$13,784
Incidence – Stroke	1.4	\$47,773
Minor Restricted Activity Days	18,563	\$1,291,673
Mortality – All Cause	26.1	\$204,506,164
Work Loss Days	3,142	\$556,370
Total		\$212,375,158