Testimony

STATE OF NEW YORK
DEPARTMENT OF HEALTH

Corning Tower The Governor Nelson A. Rockefeller Empire State Plaza Albany, New York 12237

Antonia C. Novello, M.D., M.P.H., Dr.P.H.
Commissioner

Dennis P. Whalen
Executive Deputy Commissioner

February 22, 2002

Orig-Files
C 99-F-1314
copies:
Mr. C. Patka
ALJ Harrison
ALJ Garlin
ALJ Epstein, ALJ Moynihan

RECEIVED
PUBLIC SERVICE
COMMISSION
OSEC-FILES-ALBANY
2002 FEB 22 PM 12:27

Via Hand Delivery

Honorable Janet Hand Deixler
Secretary
New York State Board on Electric Generation Siting
and the Environment
Three Empire State Plaza
Albany, NY 12223-1350

Re: Case 99-F-1314 Application of Consolidated Edison of New York, Inc. for a Certificate of Environmental Compatibility and Public Need to Repower the East River Generating Station to Replace the Waterside Generating Station in Manhattan, New York County, New York

Dear Secretary Deixler:

Please find enclosed the direct testimony of New York State Department of Health staff witness Dr. Daniel Luttinger in the above proceeding. Pursuant to 16 NYCRR 3.5(f), I have enclosed an additional five copies of this letter and the testimony.

A copy of these materials is also being provided to the Presiding and Associate Examiners as well as to the active parties to this proceeding.

Sincerely,

David W. Quist
Senior Attorney
Division of Legal Affairs
(518) 473-7978

Enclosure

cc: Honorable Robert R. Garlin (by e-mail, hard copy to follow by first-class mail)
Honorable Daniel P. O'Connell (by e-mail, hard copy to follow by first-class mail)
Active Party List (by e-mail, hard copy to follow by first-class mail)

BEFORE

THE STATE OF NEW YORK

BOARD ON ELECTRIC GENERATION SITING AND THE ENVIRONMENT

In the Matter of	:
Case 99-F-1314	:
	:
Application of Consolidated Edison	:
Company of New York, Inc. for a	:
Certificate of Environmental Compatibility	:
and Public Need to Repower its East	:
River Generating Station Located in the	:
Borough of Manhattan, New York City.	:
	:
February 22, 2002	:

Prepared Testimony of:

Daniel Luttinger, Ph.D., Chief, Toxicological Assessment Section

Bureau of Toxic Substance Assessment

New York State Department of Health

Flanigan Square, Room 330

547 River Street

Troy, New York 12180-2216

Q. Please state your name, affiliation and title.

A. I am Daniel Luttinger, Chief of the Toxicological Assessment Section in the New York State Department of Health (DOH), Bureau of Toxic Substance Assessment.

Q. Please state your employment and educational background.

A. I have been Chief of Toxicological Assessment Section since May 1998. I was Acting Chief of the Toxicological Assessment Section from November 1996 until May 1998. I joined DOH in 1993 as a research scientist in the Toxicological Assessment Section. Prior to joining DOH, I conducted research on the biological effects of chemicals in the pharmaceutical industry for approximately 11 years. I have a Bachelor of Science degree in Mathematics and a Bachelor of Science degree in Psychology, both from Carnegie-Mellon University (1975), a Ph.D. in Pharmacological and Physiological Sciences from the University of Chicago (1979) and a M.B.A. from the College of Saint Rose (1994). My resume is attached as Exhibit 1.

Q. Please state your current job responsibilities.

A. In my capacity as Chief of the Toxicological Assessment Section, I direct ten professional staff with various scientific backgrounds. My duties include health risk assessment of human exposure to chemicals; development of scientific documentation to serve as the basis for policy decisions on standards and guidelines for chemical contaminants in air, water, soil, food and consumer products; and coordination of responses to inquiries regarding the toxicity of chemicals. These responsibilities entail evaluations of toxicological and epidemiological studies on chemicals found in the environment and assessments of the human health risks associated with exposure to such chemicals.

Q. Why are you providing this testimony?

A. On January 24, 2002, the New York State Board on Electric Generation Siting and the Environment ("Siting Board") issued an order to hold an evidentiary hearing on the issue of air quality impacts of PM2.5 as it relates to the Applicant's East River Repowering Project (ERRP). To that end, the parties were directed to address a series of questions. My testimony addresses Siting Board question 2.

> What is the state of the science regarding PM2.5 and its health effects, and what is known about the general health impacts, and health risks associated with PM2.5?

In addition, my testimony provides a characterization of the potential public health impacts of emissions of PM2.5 from the ERRP based on modeling analyses conducted by the Applicant.

Q. Briefly describe your qualifications to provide testimony on the health effects of PM2.5.

A. I am familiar with the health effects of PM2.5, and other pollutants, from reviewing and commenting on the literature in the course of carrying out my DOH job responsibilities. In addition, I am the principal investigator on a study examining the effects of particulate matter and its various constituents and other air pollutants on acute asthma exacerbations.

Q. What is PM2.5?

A. PM2.5 is particulate matter with a mean aerodynamic diameter of 2.5 microns or less and is generally measured in units of mass per unit volume (e.g., micrograms per cubic meter).

Q. Can you briefly summarize the state of the science about PM2.5 and its

health effects?

A. Yes. Exposure to PM2.5 and subsequent health effects has been the subject of extensive research and integrative reviews (e.g., US EPA, 1996, US EPA, 2001, California Air Resources Board, 2001). The US EPA (2001) and California Air Resources Board (2001) documents are currently draft public documents that summarize, in part, knowledge about the health effects of PM2.5. A significant portion of this research is comprised of epidemiological studies. These studies, as summarized in the integrative reviews and reports referenced therein, have generally found associations between changes in PM2.5 concentrations in ambient (outdoor) air and changes in various health endpoints. The California Air Resources Board (2001) characterized the data on particulate matter and health effects as meeting the generally accepted guidelines for causal inferences from epidemiological studies. The health effects associated with exposure to PM2.5 include a variety of respiratory and cardiovascular endpoints, such as changes in airway peak flow measurements, alterations in heart rate variability, symptom reporting (cough, asthma, upper and lower respiratory), hospitalizations and emergency department visits for asthma, chronic obstructive pulmonary disease (COPD), and increased mortality. The elderly, children and people with lung or heart disease or asthma are among those often thought to potentially be more sensitive to exposures to airborne particles. The epidemiological studies

are fairly consistent in associating increases in ambient PM2.5 with increases in adverse health outcomes; most of the time, although not always, the increase in adverse health outcomes is statistically significant. In addition, there are a limited number of laboratory studies using animals or humans under controlled exposure conditions that link fine particles to alterations in cardiovascular and respiratory features (US EPA, 2001). The laboratory studies provide some biological plausibility for the health effects observed in the epidemiological studies.

PM2.5 is not a single chemical entity and its composition and physical characteristics vary across locations and time and may influence PM2.5 toxicity. In addition, the amounts of gaseous co-pollutants (such as sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and ozone) may also influence the association of health effects with PM2.5. Some individual components of PM2.5 have been evaluated to assess whether they can explain the toxicity of PM2.5. For instance, the World Health Organization (2000), stated that there was emerging evidence "that constituents of PM2.5, such as sulphates and strongly acidic particles, are sometimes better predictors of health effects than PM2.5." US EPA (2001) characterized recent studies that examined the role of sulfates on mortality as generally, but not always, noting an association of sulfates with mortality, and suggesting that discrepancies in the relative significance of sulfate or other components may vary across studies because of differences in their

concentrations. Similarly, epidemiological or laboratory studies have attempted to assess the potential health impacts of other components (e.g., nitrate, elemental carbon, organic carbon, metals) of PM2.5. Some of these studies suggest a greater or lesser role for a specific component of PM2.5. Definitive conclusions attributing health effects to specific components of PM2.5 are tenuous.

Q. What is the magnitude of health risks associated with increases in PM2.5?

A. A variety of risk estimates for various health endpoints have been reported for increases in PM2.5 and are described in detail in US EPA (2001). The magnitude of the estimated increase varies among studies, as do the concentration ranges of PM2.5 in the studies. The epidemiological studies to date have not identified a threshold, or a concentration below which no effects are observed. As such, at this time, a reasonable assumption is that any increase in PM2.5 exposure is associated with some increase in risk of adverse health effects.

For instance, US EPA (2001) generally estimates a 2 to 8% increase in mortality for a 25 micrograms per cubic meter increase in PM2.5. US EPA's estimates for increases in respiratory hospital admissions generally range from 5 to 15% for a 25 micrograms per cubic meter increase in

PM2.5 (US EPA, 2001).

Another way of describing or estimating the risk is illustrated in an article by Pope et al. (1995). The authors reported a correlation between median fine particulate (PM2.5) concentration and mortality rates and estimated that a yearly average increase in fine particles of 1 microgram/cubic meter was correlated with an estimate of 8 additional deaths/year/100,000 people.

Q. Are there any limitations, or uncertainties with these quantitative risk estimates?

A. Yes. Health risk estimates for any chemical or pollutant involve uncertainties. In the case of PM2.5, human health studies fairly consistently observe an association between increases in PM2.5 and health effects at concentrations generally occurring in urban environments. These factors strengthen confidence in PM2.5 risk characterizations.

Since the magnitude of the estimates of incremental increase in risk varies, and the risk estimates are based on relatively large differences in PM2.5 concentrations, uncertainty exists in extrapolating to smaller incremental

changes. Also, many of the risk estimates are calculated from studies with different background concentrations of PM2.5, different amounts of specific constituents in PM2.5, and with different air sampling methods. Any or all of these factors may contribute variability and uncertainty to the risk estimates.

Factoring other pollutants, such as gaseous pollutants (e.g., SO_2, NO_2, and ozone) into the analyses may change a risk estimate. For instance, US EPA (2001) reported that when Krewski et al. (2000) factored SO_2 into a reanalysis of Pope et al. (1995) mortality risk estimate for PM2.5, the risk estimate attributable to PM2.5 was greatly reduced. The individual epidemiological studies vary in the extent to which they control for various gaseous pollutants, which increases the variation and uncertainty in different PM2.5 risk estimates. In addition, different studies that factor gaseous pollutants into the risk estimates may observe different magnitudes of effect for those gaseous pollutants.

It may not be accurate to assume that all of the PM2.5 mass from different emission sources (e.g. burning natural gas, burning diesel fuel) is of equal toxicity. Differences in the physical/chemical characteristics (e.g., size, pH) or the specific constituents (e.g., sulfate, nitrate, elemental carbon, organic carbon, metals) of PM2.5 may influence toxicity.

The variation in risk estimates also may reflect the uncertainties around the assumed exposure used to derive the estimates. An individual's actual exposure to particulate matter, or any other air pollutant, is a function of his or her behavior and activity pattern. The use of ambient air monitoring data from a community-based monitor as a surrogate for personal exposure may over or underestimate exposure. There are indoor and localized, outdoor sources of fine particles as well as regional background concentrations, and exposure varies as people move from one microenvironment to another. The concentration of the particles, the time spent and personal activity level in each environment will influence total exposure.

Q. Is there a federal standard for PM2.5?

A. Yes. In 1996 US EPA proposed, and in 1997 established, new National Ambient Air Quality Standards (NAAQS) to regulate particulate matter. The annual PM2.5 standard is 15 micrograms per cubic meter based on the 3-year average of annual, arithmetic mean PM2.5 concentrations from single or multiple community-oriented monitors, and 65 micrograms per cubic meter, based on the 3-year average of the 98th percentile of 24-hour PM2.5 concentrations at each population-oriented monitor within an area. As noted in the Federal Register (1997) "The Act does not require the

Administrator to establish a primary NAAQS at a zero-risk level, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety."

Q. Has the Applicant estimated the annual average incremental change in PM2.5 concentrations in the area surrounding ERRP?

A. Yes, but since this is not my area of expertise I have not evaluated the modeling methods or the validity of the modeling results. The Applicant performed dispersion modeling to estimate the net changes in annual average concentrations of PM2.5, as well as PM10 (particulate matter with a mean aerodynamic diameter of 10 micrometer or less), SO_2 and NO_2 resulting from removal of the Waterside Station from service, operation of the ERRP, and Con Edison's Emissions Reduction Program. The results, for ground level and elevated receptors in Zip Codes 10002, 10003, 10009 and 10010 are presented in Tables 2-1 and 2-2 of a technical report (Mendez, 2001) that was distributed to all parties on April 12, 2001. A copy of Section 2 of that report, which presents estimates of incremental changes in concentrations to PM2.5, PM10, SO_2 and NO_2 resulting from operation of the ERRP, is attached herein as Exhibit 2.

Q. What were the results of this modeling for incremental impacts at ground-level receptors?

A. The air quality modeling results for net impacts of the ERRP indicate that annual average levels of PM2.5 will be reduced for ground-level receptor locations in the three Zip Code areas surrounding the facility. Only in Zip Code 10002 was there an increase (0.012 micrograms per cubic meter under "typical" operating conditions, and 0.056 micrograms per cubic meter under "reasonable worst case" operating conditions) in PM2.5 levels averaged over the Zip Code. In the three other Zip Codes, while there were PM2.5 increases at some locations, the spatially averaged PM2.5 levels decrease at ground level receptors by increments ranging from –0.003 to –0.035 micrograms per cubic meter.

At specific locations where PM2.5 concentrations were predicted to increase, the increase was small. Specifically, the largest estimated increase in PM2.5 at a specific location was 0.095 micrograms per cubic meter at a location in Zip Code 10002 under a "reasonable worst case" scenario. For the other three Zip Codes, the maximum estimated increase in PM2.5 ranged from 0.018 to 0.082 micrograms per cubic meter under a "reasonable worst case" scenario.

Levels of SO_2 and NO_2 were projected to decrease at all locations in all four of these Zip Codes.

Q. What were the results for incremental impacts at elevated receptor locations?

A. The estimated changes in net air impacts for elevated receptor locations (for example, tops of buildings) in the four Zip Codes are presented in Table 2-2 of the Mendez report. Under "typical" operating conditions, annual average concentrations of PM2.5, PM10, SO_2 and NO_2 are expected to decrease at all modeled receptor locations.

Under "reasonable worst case" operating conditions, annual average PM2.5, concentrations were predicted to be unchanged over all four Zip Codes, and annual average PM10, SO_2, and NO_2 concentrations were predicted to decrease over all four Zip Codes. The largest predicted increase in annual average PM2.5 concentrations, under "reasonable worst case" operating conditions, at an elevated receptor location is 0.197 micrograms per cubic meter. The annual average concentrations of SO_2 and NO_2 are expected to decrease at all elevated receptor locations.

Q. Have you drawn any conclusions based on the modeling results in Dr. Mendez's report?

A. Yes. The concentrations of PM2.5 in most cases decrease and in those cases where an increase is projected, the increase is low. Furthermore, SO_2 and NO_2 levels decrease under all scenarios. To the extent that small increases in PM2.5 would occur at some locations, the decreases in SO_2 and NO_2 may minimize any potential slight increase in risk. In addition, since PM2.5 concentrations are estimated to decrease in most areas around the community, most individuals' exposure would be less than that estimated by assuming the highest concentration is reflective of actual exposure to PM2.5 from the ERRP. Considering that the modeled impacts of PM2.5 are low and often decrease, the projected decreases in SO_2 and NO_2, and the state of the science on PM2.5 health effects, the proposed project is not likely to significantly increase risks to public health and could be determined to be compatible with public health.

Q. Does this conclude your testimony?

A. Yes.

References

California Environmental Protection Agency – Air Resources Board and Office of Environmental Health and Hazard Assessment, Review of the California Ambient Air Quality Standards for Particulate Matter and Sulfates – Public Review Draft, November 30, 2001.

Federal Register, Environmental Protection Agency 40 CFR Part 50 National Ambient Air Quality Standards for Particulate Matter; Final Rule. Vol. 62, No. 138, 38652-38701, July 18, 1997.

Krewski D, Burnett RT, Goldberg MS, Hoover K, Siemiatycki J, Jerrett M, Abrahamowicz M, White WH. Reanalysis of the Harvard Six Cities study and the American Cancer Society study of particulate air pollution and mortality. A special report of the Institute's Particle Epidemiology Reanalysis Project. Cambridge, MA: Health Effects Institute, 2000.

Mendez W. Technical Report in Support of Rebuttal Testimony of William M. Mendez, Jr. Ph.D., On behalf of Consolidated Edison Company of New York, Inc. April 12, 2001.

Pope CA III, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Heath JR CW. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Amer. J. Respir. Crit. Care Med. 1995;

151: 669-74.

US EPA, Air Quality Criteria for Particulate Matter, April 1996.

US EPA, Air Quality Criteria for Particulate Matter - Second External Review Draft, March 2001.

World Health Organization, Guidelines for Air Quality, 2000.

EXHIBIT 1

DANIEL LUTTINGER, PH.D.

CURRENT ADDRESS

Toxicological Assessment Section
New York State Department of Health
547 River St.
Troy, NY 12180-2216

(518) 402-7820 (work)

PROFESSIONAL EXPERIENCE

1993 - Present **State of New York**
Department of Health
Albany, NY 12203

Bureau of Toxic Substance Assessment

1998 Research Scientist IV
1997 Research Scientist III
1993 Research Scientist II

Major Responsibilities:

- Section Chief (11/96 – Present) responsible for assessing toxicity of chemicals, conducting health risk assessments, and recommending guidelines and standards for contaminants in air, water, soil and food.
- Design and implement a program that evaluates environmental factors related to asthma.
- Lead a team that investigates, evaluates and conducts research on the public health implications of biological contaminants.
- Design and conduct research on potential health impact of chemicals in the environment.
- Conduct physiologically based pharmacokinetic (PBPK) analyses.
- Develop toxicity profiles for specific chemicals based on a critical review of animal and human studies.
- Prepare material for legal actions.

1982 - 1993 **Sterling Winthrop Pharmaceuticals Research Division**
Rensselaer, NY 12144

Department of Neuroscience

1991 **Fellow**

1990 **Principal Research Investigator**
1986 **Group Leader**
1982 **Senior Research Biologist**

Major Responsibilities:

- Management and scientific direction of a neuropharmacology laboratory.
- Management and guidance/training of two Ph.D.'s and their electrophysiology and neurochemical laboratories.
- Identification of new projects for therapeutic targets for the treatment of CNS disorders (e.g., Alzheimer's disease, stroke, schizophrenia, depression).
- Project team leader for multidisciplinary drug discovery and drug development teams.
- CNS safety assessment for new drug candidates.
- Scientific review of potential drug candidates for in-licensing.
- Establishing collaborations with external researchers (e.g., imaging group at Mass. General Hospital).

1985 - 1997 **Albany Medical College of Union University**
Albany, NY 12208
Department of Pharmacology and Toxicology

Adjunct Associate Professor

1979 - 1982 **University of North Carolina**
Chapel Hill, NC 27514

Biological Sciences Research Center
Postdoctoral Fellow

Major Responsibilities:

- Establish behavioral testing paradigms.
- Assess potential neuroleptic activity of neurotensin.
- Publish original research and review articles.
- Write grants.
- Training and teaching undergraduates.

EDUCATION

University of Chicago, Chicago, Ill.
Ph.D. Pharmacological and Physiological Sciences
Fall, 1979

Carnegie-Mellon University, Pittsburgh, PA
B.S. Mathematics, May, 1975

B.S. Psychology, May, 1975

College of Saint Rose, Albany, NY
M.B.A., Spring, 1994

Sterling Winthrop, Inc.
Several Management Courses (e.g., frontline leadership, completed staffwork, time management)

PUBLICATIONS:

Miczek, K.A. and D. Luttinger. Differential attenuation of two kinds of conditioned suppression by d-amphetamine and pentobarbital. J. Pharmacol. Exp. Ther. 205: 282-290, 1978.

Luttinger, D. and L.S. Seiden. Increased hypothalamic norepinephrine metabolism after water deprivation in the rat. Brain Research. 208: 147-166, 1981.

Mason, G.A., C.B. Nemeroff, D. Luttinger, O.L. Hatley and A.J. Prange, Jr. Neurotensin and bombesin: differential effects on body temperature of mice after intracisternal administration. Regulatory Peptides. 1: 53-60, 1980.

Nemeroff, C.B., D. Luttinger, and A.J. Prange, Jr. Neurotensin: central nervous system effects of a neuropeptide. Trends in Neuroscience. 3: 212-215, 1980.

Luttinger, D., C.B. Nemeroff, G.A. Mason, G.D. Frye, G.R. Breese and A.J. Prange, Jr. Enhancement of ethanol-induced sedation and hypothermia by centrally administered neurotensin, beta-endorphin and bombesin. Neuropharmacology. 20: 305-309, 1981.

Nemeroff, C.B., G. Bissett, P.J. Manberg, D. Luttinger and A.J. Prange, Jr. Effects of hypothalamic peptides on the central nervous system. Peptides, Hormones and Behavior (C.B. Nemeroff and A.J. Dunn, eds.). Spectrum Publishers, New York, pp. 217-272, 1984.

Luttinger, D., C.B. Nemeroff, R.A. King, G.N. Ervin, and A.J. Prange, Jr. The effect of injection of neurotensin into the nucleus accumbens on behaviors mediated by the mesolimbic dopamine system in the rat. In: The Neurobiology of the Nucleus Accumbens (R.B. Chronister and J.R. DeFrance, eds.). Haer Institute for Electrophysiological Research Publishers, 322-332, 1981.

Heffner, T.G., D. Luttinger, J.A. Hartman and L.S. Seiden. Regional changes in brain catecholamine turnover in the rat during performance on fixed ratio and variable interval schedules of reinforcement. Brain Research. 214: 215-218, 1981.

Osbahr III, A.J., C.B. Nemeroff, D. Luttinger, G.A. Mason and A.J. Prange, Jr. Neurotensin-induced analgesia in mice: antagonism by thyrotropin releasing hormone. J. Pharmacol. Exp. Ther. 217: 645-651, 1981.

Frye, G.D., D. Luttinger, C.B. Nemeroff, R.A. Vogel, A.J. Prange, Jr. and G.R. Breese. Modification of the actions of ethanol by centrally active peptides. Peptides 2: (Suppl. 1) 99-106, 1981.

Nemeroff, C.B., D. Luttinger, D.E. Hernandez, P.W. Kalivas, E. Widerlov, G.R. Breese and A.J. Prange, Jr. Neurotensin, a peptide with certain neuroleptic-like properties. Proc. IIIrd World Congress of Biological Psychiatry, Elsevier, Amsterdam. pp. 363-367, 1982.

Luttinger, D., C.B. Nemeroff and A.J. Prange, Jr. The effects of neuropeptides on discrete-trial conditioned avoidance responding. Brain Research. 237: 183-192, 1982.

Nemeroff, C.B., A.J. Prange, Jr., D. Luttinger, D.E. Hernandez, R.A. King and S.K. Burgess. Similarities and differences in the effects of centrally administered neurotensin and neuroleptics. Pschopharmacology Bulletin 17: 145-147, 1981.

Nemeroff, C.B., D. Luttinger and A.J. Prange, Jr. Neurotensin and Bombesin. In: Handbook of Psychopharmacology, Vol. 16 (L.L. Iversen, S.D. Iversen and S.H. Snyder, eds.). Plenum Press, New York. pp. 363-466, 1983.

Burgess, S.K., D. Luttinger, D. Hernandez, P.W. Kalivas, C.B. Nemeroff and A.J. Prange, Jr. Antagonism of the Central Nervous System Effects of Neurotensin by Thyrotropin Releasing Hormone. In: Thyrotropin Releasing Hormone. (E.C. Griffiths and G.W. Bennett, eds.). Raven Press, New York. pp. 291-302, 1983.

Nemeroff, C.B. and D. Luttinger. The Anhedonia Hypothesis of Neuroleptic Drug Action: Basic and Clinical Considerations. The Behavioral and Brain Sciences. 5: 70-71, 1982.

Luttinger, D., G.D. Frye, C.B. Nemeroff and A.J. Prange, Jr. The effects of neurotensin, beta-endorphin and bombesin on ethanol-induced behaviors in mice. Psychopharm. 79: 357-363, 1983.

Luttinger, D., R.A. King, D. Sheppard, J. Strupp, C.B. Nemeroff and A.J. Prange, Jr. The effect of neurotensin on food consumption in the rat. Eur. J. Pharmacol. 81: 499-504, 1982.

Luttinger, D., G.D. Frey and G. Bissette. Effects of neurotensin on the actions of barbiturates and ethanol. Ann. N.Y. Acad. Sci. 400: 259-266, 1982.

Bissette, G., D. Luttinger, G.A. Mason, D.E. Hernandez and P.T. Loosen. Neurotensin and thermoregulation. Ann. N.Y. Acad. Sci. 400: 268-281, 1982.

Nemeroff, C.B., D.E. Hernandez, D. Luttinger, P.W. Kalivas and A.J. Prange, Jr. Interactions of neurotensin with brain dopamine systems. Ann. N.Y. Acad. Sci. 400: 330-342, 1982.

Luttinger, D., S.K. Burgess, C.B. Nemeroff and A.J. Prange, Jr. The effects of chronic morphine treatment on neurotensin-induced antinociception. Psychopharm. 81: 10-13, 1983.

Nemeroff, C.B., D. Luttinger, D.E. Hernandez, R.B. Mailman, G.A. Mason, S.D.Davis, E.

Widerlov, G.D. Frey, C.A. Kilts, K. Beaumont, G.R. Breese and A.J. Prange, Jr. Interactions of neurotensin with brain dopamine systems: biochemical and behavioral studies. J. Pharmacol. Exp. Therap. 225: 337-345, 1983.

Luttinger, D., D.E. Hernandez, C.B. Nemeroff and A.J. Prange, Jr. Peptides and nociception International Review of Neurobiology. 25: 185-241, 1984.
Hlasta, D.J., D.R. Haubrich and D. Luttinger. Antidepressants. In: CRC Handbook of Medicinal Chemistry, M. Verderame, ed. pp. 277-302, 1986.

Luttinger, D., R. Ferrari, M. Perrone and D.R. Haubrich. Pharmacological analysis of alpha-2-adrenergic mechanisms in nociception and ataxia. J. Pharmacol. Exp. Ther. 232, 883-889, 1985.

Luttinger, D. and M.E. Durivage. Alpha-2-adrenergic antagonists effect on amphentamine-induced behaviors. Pharm. Biochem. Behav. 25: 155-160, 1986.

Luttinger, D., M. Freedman, L. Hamel, S.J. Ward and M. Perrone. The effects on serotonin antagonists in a behavioral despair procedure in mice. Eur. J. Pharmacol. 107: 53-58, 1985.

Luttinger, D. Determination of drug efficacy in mice by use of different water temperatures in a tail-immersion test. J. Pharm. Meth., 13: 351-357, 1985.

Mason, P., J. Skinner and D. Luttinger. Two tests in rats for antianxiety effects of clinically anxiety-attentuating antidepressants. Psychopharm. 92: 30-34, 1987.

Merrit, W.D., G. Bissette, D. Luttinger, A.J. Prange, Jr. and C.B. Nemeroff. Adaptation to cold antagonizes neurotensin-induced hypothermia in mice. Brain Res. 369: 136-142, 1986.

Baizman, E.R., A.M. Ezrin, R.A. Ferrari and D. Luttinger. Pharmacological Profile of Fezolamine Fumarate: A Non-Tricyclic Antidepressant in Animal Models. J. Pharm. Exp. Ther. 243: 40-54, 1987.

Wentland, M.P., D.M. Bailey, E.J. Alexander, M.J. Castaldi, R.A. Ferrari, D.R. Haubrich, D.A. Luttinger and M.H. Perrone. The synthesis and pharmacological properties of novel 2-substituted-4,5-dihydro-1-H-imidazole derivatives. J. Med. Chem. 30: 1482-1489, 1987.

Hlasta, D.J., D. Luttinger, M.H. Perrone, M.J. Silbernagle, S.J. Ward and D.R. Haubrich. Alpha-2-adrenergic Agonists/Antagonists: The synthesis and structure-activity relationships of a series of indolin-2-yl and tetrahydroquinolin-2-yl imidazolines. J. Med. Chem. 30: 1555-1562, 1987.

Luttinger, D. and D.J. Hlasta. Antidepressant Agents. In: Annual Reports in Medicinal Chemistry, Vol. 22, edited by D. Bailey, Academic Press, Orlando, pp. 21-30, 1987.

Herrick-Davis, K., S. Chippari, D. Luttinger and S.J. Ward. Evaluation of Adenosine Agonists as Potential Analgesics. Eur. J. Pharmacol. 162: 365-369, 1989.

Rossitch, E., Jr., R.A. King, D. Luttinger and C.B. Nemeroff. Behavioral Effects of Neurotensin: Operant Responding and Assessment of Anhedonia. Eur. J. Pharmacol. 163: 119-122, 1989.

Perrone, M.H., D. Luttinger, L.T. Hamel, P.M. Fritz, R. Ferraino and D.R. Haubrich. In Vivo Assessment of Napamezole, An Alpha-2-Adrenoceptor antagonist and Monamine Reuptake Inhibitor. J. Pharm. Exp. Ther. 254: 476-483, 1990.

Haubrich, D.R., S.J. Ward, E. Baizman, M.R. Bell, J. Bradford, R. Ferrari, M. Miller, M. Perrone, A.K. Pierson, J.K. Saelens and D. Luttinger. Pharmacology of Pravadoline: A Novel Antinociceptive Agent. J. Pharm. Exp. Ther. 255: 511-522, 1990.

Bell, M.R., T.E. D'Ambra, V. Kumar, M.A. Eissenstat, J.L. Herrmann, Jr., J.R. Wetzel, D. Rosi, R.E. Philion, S.J. Daum, D.J. Hlasta, R.K. Kullnig, J.A. Ackerman, D.R. Haubrich, D.A. Luttinger, E.R. Baizman, M.S. Miller and S.J. Ward. Antinociceptive aminoalkylindoles. J. Med. Chem. 34: 1099-1110, 1991.

Fritz, P.M., D.L. DeHaven-Hudkins, L.C. Fleissner, F.Y. Ford-Rice and D. Luttinger. Sigma binding affinity and inhibition of apomorphine-induced climbing. Eur. J. Pharmacol. 235: 229-235, 1993.

Lo, E.H., K. Matsumoto, A.R. Pierce, L. Garrido and D. Luttinger. Pharmacologic reversal of acute changes in diffusion-weighted magnetic resonance imaging in focal cerebral ischemia. J. Cerebral Blood Flow and Metabolism. 14: 597-603, 1994.

Fritz, P.M., G. Recer, and D. Luttinger. Asthma Among Adult New Yorkers. Behavioral Risk Factor Surveillance System 7(No. 1), 1-4, 1999

Boss, L.P., R.A. Kreutzer, D. Luttinger, J. Leighton, K. Wilcox and S.C. Redd. The Public Health Surveillance of Asthma. Journal of Asthma 38(1), 83-89, 2001.

PUBLISHED ABSTRACTS:

Heffner, T.G., D. Luttinger, J.A. Hartman and L.S. Seiden. Regional changes in CNS catecholamine metabolism during performance on fixed ratio or variable interval operant schedules. Neuroscience. 5: 1112, 1979.

Luttinger, D., G.A. Mason, G.D. Frye, A.J. Osbahr, III, C.B. Nemeroff and A.J. Prange, Jr. Potentiation of ethanol-induced narcosis and hypothermia by neurotensin. Transactions of the American Society for Neurochemistry. 11: 355, 1980.

Osbahr III, A.J., C.B. Nemeroff, D. Luttinger, G.A. Mason and A.J. Prange, Jr. The effects of naloxone and thyrotropin-releasing hormone (TRH) on neurotensin (NT)-induced analgesia in mice. Clinical Research 28: 241-A, 1980.

Nemeroff, C.B., D. Luttinger, G.A. Mason, G.D. Frye, G.R. Breese and A.J. Prange, Jr. Effects of intracisternal neurotensin, beta-endorphin, or bombesin on ethanol-induced sedation and hypothermia. Fifth Biennial International Symposium on Alcoholism, June 9-13, 1980, Cardiff, Wales, Drug and Alcohol Dependence. 6: 118, 1980.

Nemeroff, C.B., D. Luttinger, A.J. Osbahr, III, G. Bissette and A.J. Prange, Jr. Neurotensin and thyrotropin-releasing hormone (TRH) are "antagonists" in several behavioral and pharmacological paradigms. International Society of Psychoneuroendocrinology, Eleventh Annual Meeting, 1980, Florence, Italy 107.

Luttinger, D., R. Wiggins, R.A. King, C.B. Nemeroff and A.J. Prange, Jr. Inhibition of conditioned advoidance responding and rewarding elecrical self-stimulation of the brain by centrally administered neurotensin. Neurosciences. 6: 283.7, 1980.

Nemeroff, C.B., D. Luttinger, A.J. Osbahr, III and A.J. Prange, Jr. Neurotensin blocks behaviors induced by indirect, but not direct dopamine agonists: evidence for mediation at a mesolimbic dopamine termination site, the nucleus accumbens. Neuroscience. 6: 283.6, 1980.

Frye, G.D., D. Luttinger, C.B. Nemeroff, R. Vogel, A.J. Prange, Jr. and G.R. Breese. Modification of the actions of ethanol by centrally active peptides. Proc. Winter Neuropeptide Conference, Breckenridge, Colorado, January 21-24, 1981.

Luttinger, D., C.B. Nemeroff and A.J. Prange, Jr. Investigations of the interactions of ethanol with neuropeptides. In: Alcoholism: Clinical and Experimental Research, 1981.

Nemeroff, C.B., D. Luttinger and A.J. Prange, Jr. Blockage of neurotensin-induced antinociception by thyrotropin-releasing hormone in mice. XII. Congress of the Intl. Soc. of Psychoneuroendocrinology, Montreal, Quebec, May 25-28, 1981.

Luttinger, D., C.B. Nemeroff and A.J. Prange, Jr. Inhibition of neurotensin-induced antinociception in morphine tolerant mice. The Pharmacologist. 23: 120, 1981.
Nemeroff, C.B., D. Luttinger, D.E. Hernandez, E. Widerlov, G.R. Breese and A.J. Prange, Jr. Neurotensin, a peptide with certain neuroleptic-like properties. Third World Congress of

Biological Psychiatry. June 28 - July 3, 1981.

Bisette, G., D. Luttinger, C.B. Nemeroff and A.J. Prange, Jr. The effects of d-amphetamine, methylphenidate and apomorphine on neurotensin-induced hypothermia in mice. Neuroscience. 7: 32, 1981.

Jennes, L., W.E. Stumpf, W.C. Beckman, S.K. Burgess, D. Luttinger, C.B. Nemeroff and A.J. Prange, Jr. Neurotensin immunoreactive systems in the central nervous system of the rat. Neuroscience. 7: 223, 1981.

Luttinger, D., D. E. Hernandez, R.B. Mailman, C.B. Nemeroff and A.J. Prange, Jr. Interactions of neurotensin with direct and indirect dopamine agonists: neurochemical and behavioral measures. Neuroscience. 8: 309, 1982.

Bisette, G., D. Luttinger, C.B. Nemeroff, V.M. Miller and A.J. Prange, Jr. Regional brain distribution of neurotensin-like immunoreactivity in the woodchuck (Marmota Monak). Neuroscience. 8: 584, 1982.

Merritt, W.D., G. Bissette, D. Luttinger, A.J. Prange, Jr. and C.B. Nemeroff. Prior cold exposure antagonizes neurotensin-induced hypothermia in mice. Neuroscience. 9: 141, 1983.

Luttinger, D. The effects of different water temperatures on the antinociceptive action of various analgesics in a tail-immersion test in mice. IVth World Congress on Pain of the International Association for the Study of Pain. Pain Suppl. 2: S159, 1985.

Luttinger, D. and M.E. Durivage. The effects of alpha-2-antagonists on amphetamine-induced increases in locomotor activity and stereotypy. Neuroscience. 10: 1176, 1984.

Luttinger, D. and P.A. Mason. The effects of antidepressants, which attenuate clinical anxiety associated with depression, in two antianxiety procedures in rats. Neuroscience. 11: 184, 1985.

Bailey, D.M., E.J. Alexander, M.J. Castaldi, R.A. Ferrari, D.R. Haubrich, D.A. Luttinger, M.H. Perrone and M.P. Wentland. The synthesis and pharmacological activity of a series of 2-[(2-naphthalenyl)methyl]-4,5-dihydro-1H-imidazole derivatives. 191st National American Chemical Society National Meeting, 57, 1986.

Luttinger, D. and S.M. Patton. The effects of adenosine agonists on apomorphine-induced climbing in mice. Neuroscience. 12: 911, 1986.

Haubrich, D.R., E.R. Baizman, M.R. Bell, M. Miller, A.K. Pierson, J.K. Saelens, V.J. Stecher, S.J. Ward and D. Luttinger. Antinociceptive activity of Win 48098. The Pharmacologist. 28: 140, 1986.

Luttinger, D., D.R. Haubrich, L.T. Hamel, P.M. Fritz and M.H. Perrone. Napamezole: An alpha-2 adrenergic antagonist that inhibits monoamine reuptake. The Pharmacologist. 28: 90, 1986.

Perrone, M.H., L.T. Hamel, D. Luttinger, D.M. Bailey, R.A. Ferrari and D.R. Haubrich. Napamezole: an alpha-2 adrenergic antagonist. The Pharmacologist. 28: 90, 1986.

Hlasta, D.J., D.R. Haubrich, D.A. Luttinger, M.H. Perrone, M.J. Silbernagel and S.W. Ward. 2-(4,5-Dihydro-1H-imidazol-1-yl)-2,3-dihydro-1H-indoles: novel alpha-2-adrenergic agonists and antagonists. 192nd ACS National Meeting, Abstract No. 17, 1986.

Luttinger, D., R.A. Ferrari, M.H. Perrone and D.R. Haubrich. Pharmacological analysis of alpha-2 adrenergic mechanisms in nociception and ataxia. Twentieth National Medicinal Chemistry Symposium, 1986.

Baizman, E.R., M.R. Bell, J.C. Bradford, R.A. Ferrari, D.R. Haubrich, D. Luttinger, M.S. Miller, M.H. Perrone, A.K. Pierson, J.K. Saelens and S.J. Ward. Antinociceptive activity of Win 48098. British Pharmacological Society, 90: 66P, 1987.

Herrick, K., S.M. Patton, D. Luttinger and S.J. Ward. Evaluation of adenosine agonists as potential analgesics. Federation Proceedings, 46: 382, 1987.

Ward, S.J., D. Luttinger, E.R. Baizman, B.M. Berger, J.C. Bradford, G. Grieco, D.R. Haubrich, M.S. Miller, M.H. Perrone, A. Pierson, J.K. Saelens, J.B. Skinner and R. Stroshane. Pharmacologic Profile of Win 48098, a New Analgesic. Vth World Congress on Pain of the International Association for the Study of Pain. Pain Suppl. 4: S47, 1987.

Fritz, P.M. and D. Luttinger. The effects of adenosine agonists on discrete trial avoidance behavior in rats. Neuroscience. 13: 999, 1987.

Bell, M.R., T.E. D'Ambra, V. Kumar, M.A. Eissenstat, D.J. Hlasta, D. Rosi, S.J. Ward, A.K. Pierson, D.R. Haubrich, J.K. Saelens, D. Luttinger, M.S. Miller, E.R. Baizman, M.H. Perrone, J.F. Baker and M.P. Zabinski. Antinociceptive aminoalkylindoles: structure activity relationships and possible mode of action. 194th ACS National Meeting, New Orleans, LA, Aug. 30-Sept. 4, 1987, MEDI ABSTRACT 19.

Baizman, E., N. Beglin, D. Koonz and D. Luttinger. Inhibition of NSAID - induced antinociception in mice by alpha-2 adrenergic receptor blockade. Neuroscience. 14: 851, 1988.

Ward, S.J., M. Miller, D. Luttinger, M.A. Eissenstat and M. Bell. Inhibitory activity of analogs of Win 48098 in isolated tissue preparations In Vitro is reflective of a mechanism of antinociception. Neuroscience. 14: 324, 1988.

Luttinger, D., D. Koonz and S. Chippari. Phenyl-P-Quinone Writhing in Mice: Can it predict analgesic efficacy? The Pharmacologist. 30: A155, 1988.

Ward, S.J., E. Baizman, M. Bell, S. Childers, T. D'Ambra, M. Eisenstate, K. Estep, D. Haycock,

A. Howlett, D. Luttinger, M. Miller and M. Pacheo. Aminoalkylindoles (AAIs): A new route to the canabinoid receptor? Comm. Prob. Drug Depend., NIDA Research Monograph. 105: 405-408, 1990.

Casiano, F.M., R. Arnold, M. Bell, S. Childers, M. Eissenstat, K. Estep, D. Haycock, D. Luttinger, M. Pacheo and S.J. Ward. Putative aminoalkylindole (AAI) antagonists. Comm. Prob. Drug Depend., 1990.

Luttinger, D., E.R. Baizman, M.S. Miller and S.J. Ward. The in vivo effects of aminoalkylindoles, a new class of compounds which interact with a cannabinoid binding site. Comm. Prob. Drug Depend., 1990.

Luttinger, D. and D. Koonz. Comparison of the behavioral effects of competitive and non-competitive NMDA antagonists in mice. Neuroscience. 16: 858, 1990.

Miller, M.S., P.M. Fritz, M. Lewis and D. Luttinger. NMDA-Induced fodrin turnover in vivo: association with neuronal injury and dissociation from convlusions. Neuroscience. 18: 440, 1992.

Miller, M.S., B. Ault, D.L. DeHaven-Hudkins, W. Earley, D. Luttinger, J. Mallamo and S. Ward. Win 63480: A novel open channel - selective antagonist with antischemic activity. Neuroscience 20: 1994.

Ault B., D. Luttinger, S.J. Ward, J.P. Mallamo, D. Martin, T.E. Salt, S.A. Eaton, X. Zheng, R.S. Zukin, and M.S. Miller. Win 63480-2, A high affinity uncompetitive NMDA channel blocker, has reduced closed channel access compared to MK-801. Neuroscience 20: 1994.

Luttinger, D., P.M. Fritz, G.M. Recer, C.M. Maylahn, and E.L. Dones. Asthma among NYS Minorities. Working Together to Combat Urban Asthma (New York Academy of Medicine), 1998.

Luttinger D., and P. Fritz. Use of the Behavioral Risk Factor Surveillance System (BRFSS) for Assessing Information about Adult Asthma in New York State. Centers for Disease Control and Prevention – A Public Health Response to Asthma Conference, 1999.

Bogdan K.G., A.J. Grey, and D.L. Luttinger. An Ambient Air Guideline for Tetrachloroethene based on Non-Oncogenic Effects in Humans. Society of Toxicology Annual Meeting, 1999.

Luttinger, D. Hazardous Air Pollutants and Acute Asthma in Urban Areas. NYSERDA - Environmental Monitoring, Evaluation and Protection in New York: Linking Science and Policy, 1999.

Recer, G., P. Fritz, C. Maylahn and D. Luttinger. Asthma Among New York State Minorities. Centers for Disease Control and Prevention - A Public Health Response to Asthma Conference, 2000.

Bogdan, K.G. and D. Luttinger. A Cancer Potency Factor for Evaluating Exposure to Low-

Level Oral MTBE Exposures. 32nd Mid-Atlantic Industrial and Hazardous Waste Conference, 2000.

Bogdan, K.G. and D. Luttinger. A Cancer Potency Factor for Evaluating Low-Level Oral MTBE Exposures. Society of Toxicology Annual Meeting, 2001.

Bari, A., V. Ferraro, V.A. Dutkiewicz, C.D. Judd, L.R. Wilson, D.A. Luttinger, and L. Husain. Measurements of Gaseous Nitrous Oxide, Nitric Acid, Sulfur Dioxide, Hydrochloric Acid and Particulate Sulfate and PM(2.5) in New York. Submitted American Assoc. Aerosol Research, 2001.

Luttinger, D. and L. Wilson. New York City Asthma Study. NYSERDA - Environmental Monitoring, Evaluation and Protection in New York: Linking Science and Policy, 2001.

EXHIBIT 2

State of New York
The New York State Board of Electric
Generation Siting and the Environment

In the Matter of the Application of Consolidated Edison Company of New York, Inc. for a Certificate of Environmental Compatibility and Public Need Pursuant to Article X of the New York State Public Service Law to Repower its East River Generating Station in Manhattan, New York County, New York	**Case No. 99-F-1314**

TECHNICAL REPORT
IN SUPPORT OF REBUTTAL TESTIMONY OF
WILLIAM M. MENDEZ, Jr., Ph.D

On Behalf of
Consolidated Edison Company of New York, Inc.

April 12, 2001

2. CHANGES IN EXPOSURES TO CRITERIA POLLUTANTS RESULTING FROM OPERATION OF THE ERRP

It is fundamental to the estimation of potential health impacts of the ERRP to understand the changes in exposures to pollutants that would be associated with the ERRP operations. As noted by the project engineers, in the Supplemental Direct Testimony of Stephen A. Kurtz and the emissions report annexed as Kurtz Exhibit 1 (Kurtz 2001), upgrades to the East River Plant will result in projected increases in emissions of particulate matter and CO, and projected reductions in SO_2 and NO_2 from the facility itself. However, aggregate criteria pollutant emissions from the Con Edison Steam System are projected to be reduced, and the total emissions of the individual criteria pollutants are projected to decrease as a result of the shutdown of the Waterside Plant and shifts in load distribution among the remaining plants.

These changes in locations and amounts of emissions for Con Edison facilities result in changes in the patterns of exposures, not only in the vicinity of the ERRP itself, but also in the rest of New York City (Slack 2001). The intervenors maintain that the area of most concern is Zip Code 10009 in general, and the inhabitants of certain high-rise buildings near the ERRP in particular. A review of the net air quality impact modeling results (Slack 2001) shows that this view is not correct, and that air quality changes associated with the ERRP are widespread, and primarily positive, across New York City (and the suburbs). As will be discussed in more detail

below, areas most affected by changes in emission include not just parts of Zip Code 10009, but also most of Zip Code 10002 (which actually experiences the largest changes), a large part of Zip Code 10010, and part of Zip Code 10003.

2.1 Changes in Ground-Level Exposures

We begin with a discussion of changes in ground-level exposures in the Zip Codes most affected by the ERRP because these exposures are representative of the changes in ambient (outdoor) air concentrations experienced by the large majority of residential receptors for the large majority of the time. Changes in exposure levels at elevated receptors will be discussed in the following section.

As will be seen below, Zip Code 10009 is not the only area affected by ERRP emissions. The ERRP is located near the northern boundary of Zip Code 10009, just south of Zip Code 10010. Zip Code 10003 is located to the west of Zip Code 10009, and Zip Code 10002 is located to the south. Average and maximum changes in exposure have been calculated for these zip codes.

Table 2-1 shows the estimated net changes in annual average ground-level exposures to PM2.5, PM10, SO_2 and NO_2 associated with the ERRP in these four Zip Codes. A more detailed discussion of the air modeling results are provided in Mr. Slack's testimony (Slack, 2001). The values shown are the estimated average change in ground-level concentration in each Zip Code, based on all the receptor locations[3], and the range of concentration changes from the lowest to highest receptor locations. A positive value in the "average" column indicates that the average concentration among all receptor locations in the Zip Code is projected to increase, while a negative number in the "average" column indicates that the average concentration is projected to decrease. These net changes include both concentration increments resulting from the ERRP and concentration decreases resulting from closing down the Waterside plant. They represent a comparison, to the best approximation, of the difference between exposures if the Steam System continued to operate as it did in recent years, and what is expected to occur when the ERRP comes on line. These are therefore the appropriate values to use in judging the potential for adverse (or positive) health impacts from the project. Projections are made for two ERRP emissions scenarios (Slack 2001), corresponding approximately to typical and reasonable worst-case combinations of load and fuel mixtures.

Table 2-1. Net Changes in Ground-Level Exposures Associated with ERRP Operation

Zip Code 10002	**Emissions Scenario 1**		**Emissions Scenario 2**	
Pollutant	**Average (ug/M3)**	**Range (ug/M3)**	**Average (ug/M3)**	**Range (ug/M3)**
PM2.5	0.012	-0.014 to 0.027	0.056	0.001 to 0.095
PM10	0.000	-0.033 to 0.014	0.045	-0.005 to 0.077
SO2	-0.453	-1.21 to -0.205	-0.438	-1.09 to -0.197
NO2	-0.636	-1.34 to -0.330	-0.556	-1.19 to -0.288
Zip Code 10003	**Emissions Scenario 1**		**Emissions Scenario 2**	
Pollutant	**Average (ug/M3)**	**Range (ug/M3)**	**Average (ug/M3)**	**Range (ug/M3)**

[3] Since the receptor grids in the Zip Codes are uniformly distributed, these values do not represent population-weighted averages.

PM2.5	-0.033	-0.056 to -0.007	-0.022	-0.040 to 0.018
PM10	-0.037	-0.063 to -0.010	-0.026	-0.046 to 0.009
SO2	-0.162	-0.384 to -0.348	-0.158	-0.373 to -0.074
NO2	-0.620	-1.02 to -0.259	-0.600	-0.986 to -0.243
Zip Code 10009	**Emissions Scenario 1**		**Emissions Scenario 2**	
Pollutant	**Average (ug/M3)**	**Range (ug/M3)**	**Average (ug/M3)**	**Range (ug/M3)**
PM2.5	-0.035	-0.091 to 0.001	-0.008	-0.072 to 0.082
PM10	-0.049	-0.125 to -0.017	-0.022	-0.091 to 0.055
SO2	-0.567	-1.57 to -0.011	-0.556	-1.55 to -0.011
NO2	-0.843	-1.78 to -0.241	-0.794	-1.67 to -0.241
Zip Code 10010	**Emissions Scenario 1**		**Emissions Scenario 2**	
Pollutant	**Average (ug/M3)**	**Range (ug/M3)**	**Average (ug/M3)**	**Range (ug/M3)**
PM2.5	-0.027	-0.074 to -0.001	-0.003	-0.041 to 0.048
PM10	-0.036	-0.099 to -0.003	-0.012	-0.065 to 0.030
SO2	-0.348	-0.972 to -0.075	-0.338	-0.952 to -0.072
NO2	-0.757	-1.34 to -0.182	-0.713	-1.27 to -0.165

The first important feature of these results is the overwhelming predominance of negative values (decreasing exposures), even for particulates. Operation of the ERRP is projected to result in decreases in exposures to most pollutants at the overwhelming majority of locations in these four Zip Codes. This is no surprise in the cases of SO_2 and NO_2, since emissions from the ERRP would be lower than current emissions from the East River Plant. What is perhaps unexpected, however, it that the estimated PM2.5 levels, whose emissions from the East River Plant would increase, also decrease at most locations. Only in Zip Code 10002 does the spatially averaged estimated PM2.5 exposure increase as a result of the ERRP. In the three other Zip Codes, while PM2.5 levels increase at some locations, the average ground-level PM2.5 levels decrease under both emission scenarios. This occurs because decreases in emissions from the Waterside Plant offset increased emissions from the ERRP. Average exposures to PM10 likewise increase only in Zip Code 10002, while exposures to SO_2 and NO_2 decrease at every receptor location in all four Zip Codes.

Another key feature of the data is the small magnitude of particulate level increases, where they do occur. Recall that in Section 1 of this report, estimated changes in projected health effects of pollutants derived from epidemiological studies were expressed in terms of increase per 10 ug/M^3 or per 50 ug/M^3 in exposures. In Table 2-1, the largest increase in average PM2.5 exposure at any location is 0.095 ug/M^3, which represents the maximum increase in exposure in Zip Code 10002. The maximum increment in the other three Zip Codes ranges from 0.018 to 0.082 ug/M^3. The increase in <u>average</u> PM2.5 exposure in Zip Code 10002 (the average exposure decreases in the other Zip Codes) is just 0.056 ug/M^3.

While it is theoretically possible that localized increases in average particulate exposures of this magnitude might be associated with some increase in risk of adverse health effects, it is not likely that this increase could be identified in any meaningfully sized population. This is both because of the inherent uncertainty associated with exposure-response assessment for particulates,

and because normal population mobility (moving between home, school, work, shopping, or other activities) and behavior (smoking, opening windows, turning on air conditioning) would "wipe out" the effects of small changes in ambient concentration. That would also be true for day-to-day variations in pollutant exposure, which would be somewhat higher than the variations in long-term average values.

The final important feature of the data is the relatively large decreases in exposures to the gaseous pollutants (SO_2 and NO_2) at locations where exposures to particulates are increased. In Zip Code 10002, where the average exposure to PM2.5 increases by 0.056 ug/M^3, average exposures to SO_2 and NO_2 are decreased by 0.438 and 0.556 ug/M^3, respectively. That is, the decrease in SO_2 is about nine times greater than the increase in PM2.5, and the decrease in NO_2 levels is about 10 times the PM2.5 increase. Similarly, the largest decreases in SO_2 and NO_2 exposures in the four Zip Codes are much larger (at least 10-fold) than the largest increases in PM2.5 and PM10 exposures. (It is fair to note, however, that the increases and decreases do not occur at the same locations, so they are not precisely offsetting.) Thus, even if it were argued that the small localized increases in PM exposures were meaningful from a health standpoint, it would also have to be admitted that the much larger decreases in SO_2 and NO_2 should also be considered, and would have offsetting, positive impacts on health. This is particularly the case, since as reviewed in Section 1, the health effects of PM, SO_2 and NO_2 are similar in character, if not in magnitude. While it is not possible to estimate the magnitudes of the health effects of small changes in particulate exposures, the presence of the large offsetting decreases in exposure to gaseous pollutants is a strong reason why the ERRP emissions will not have a net decremental impact on public health.

2.2 Changes in Exposure at Elevated Receptors

Changes in net air impacts associated with the ERRP were also calculated for selected elevated receptor locations (Slack 2001). As noted therein, these locations do not represent locations where populations, or even individuals, would be exposed to pollutants. Rather they represent the tops of man-made structures near the ERRP where air pollution impacts were expected to be the highest. The net air quality impacts for the elevated receptors are summarized in Table 2-2. The numbers in the "Range" column represent the lowest and highest of the average concentrations for elevated receptors in each of the four ZIP Codes (10002, 10003, 10009, 10010).

Table 2.2. Net Changes in Air Quality Associated with the ERRP at Elevated Receptors

Pollutant	Emissions Scenario 1		Emissions Scenario 2	
	Average, ug/M^3	Range, ug/M^3	Average, ug/M^3	Range, ug/M^3
PM2.5	-0.043	-0.164 to -0.004	0.000	-0.079 to 0.197
PM10	-0.065	-0.235 to -0.015	-0.022	-0.149 to 0.123
SO_2	-0.917	-3.32 to -0.199	-0.901	-3.243 to -0.196
NO_2	-1.202	-3.95 to -0.439	-1.125	-3.796 to -0.427

It can be seen that, even at these locations whose concentration levels are most affected by the ERRP, average levels of all the pollutants are expected to decrease at the elevated receptors

under emissions Scenario 1. In the case of Scenario 2, average PM2.5 levels are predicted to remain unchanged within the limits of the air quality model, and average levels of all the other pollutants are expected to decrease. The largest predicted increase in long-term average PM2.5 levels at any elevated receptor is 0.197 ug/M^3, and the increase in PM10 is 0.123 ug/M^3. As was the case for the ground-level receptors, projected average decreases in SO_2 and NO_2 are substantially larger than the decreases in other pollutants.

2.3 Summary of Net Air Impacts Analysis

Air quality modeling of the net impacts of the ERRP (increases from the ERRP and decreases from other plants) show that long-term average levels of PM2.5, PM10, SO_2 and NO_2 will all be reduced for the large majority of ground-level receptor locations under both emissions scenarios. The concentrations of SO_2 and NO_2 are reduced at all ground-level receptors at all locations under both scenarios. Where increases in estimated levels of PM2.5 and PM10 occur, they are offset by substantially larger decreases in SO_2 and NO_2. Even at elevated receptors near the ERRP, increases in PM levels are small and offset by reductions in the levels of SO_2 and NO_2. These findings indicate that the vast majority of New York City residents will experience improved air quality as a result of the ERRP, and suggest that even in the very circumscribed areas where increases in PM exposure may occur, the potential for adverse health effects is quite small. As noted in Section 1, exposures to residents in individual buildings are primarily determined by factors other than ambient PM levels. This fact, combined with small increase in estimated PM exposure and the large decreases in SO_2 and NO_2 levels, strongly suggest that no significant adverse effect will occur among the residents of Haven Plaza.